Part 3

SYNTHESIS

Population Ecology: A Unified Study of Animals and Plants Michael Begon, Martin Mortimer and David J. Thompson
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Chapter 6

Population regulation

6.1 Introduction

There have, in the past, been contrasting theories to explain the abundance of animal and plant populations. Indeed, the interest has been so great, and the disagreement often so marked, that the subject has been a dominant focus of attention in population ecology throughout much of this century. The present view, however, is that the controversy has not so much been resolved, as recognized for what it really is (like so many other controversies): a product of the protagonists taking up extreme positions and arguing at cross purposes. In this chapter we shall confine ourselves to a brief summary of the main arguments in the controversy (sections 6.2–6.5). This should provide the necessary background, but will avoid our making too much use of historical hindsight. It would be unwise (and unfair) to analyse the arguments in too great a detail, since most were propounded between 1933 and 1958 when understanding of population ecology was even less sophisticated than it is now. More inclusive reviews may be found in Bakker (1964), Clark *et al.* (1967), Klomp (1964) and Solomon (1964). In section 6.6 we shall illustrate how the problem seemed to have been resolved by the extensive life table analysis of a Colorado beetle population. In section 6.7 we shall look at the resurrection of the problem in the 1980s, by exposing just what life table analyses do not tell us, particularly with respect to spatial density-dependence. We shall end this section by suggesting how life-table data will have to be collected in the future, and report some encouraging results from the most extensive set of census data yet published, which reaffirms the importance of density-dependent population regulation. Then, in sections 6.8–6.11, we shall deal with a heterogeneous sequence of topics, all of which have a bearing on the general question of how population sizes are regulated and determined.

6.2 Nicholson's view

A.J. Nicholson, an Australian ecologist, is usually credited as the major proponent of the view that density-dependent, biotic interactions (which Nicholson called 'density-governing reactions') play the main role in determining population size (Nicholson, 1933, 1954a, b, 1957, 1958). In his own words: 'Governing reaction induced by density change holds populations in a state of balance in their environments', and '... the mechanism of density governance is almost always intraspecific competition, either amongst the animals for a critically important requisite, or amongst natural enemies for which the animals concerned are requisites'. Moreover, although he recognized that '... factors which are uninfluenced by density may produce profound effects upon density', he considered that they only did so by '... modifying the properties of the animals, or those of their environments, so influencing the level at which governing reaction adjusts population densities'. Even under the extreme influence of density-independent factors '... density governance is merely relaxed from time to time and subsequently resumed, and it remains the influence which adjusts population densities in relation to environmental favourability' (all Nicholson 1954b). In other words, Nicholson may be taken to represent the view that density-dependent processes '... play a key role in the determination of population numbers by operating as stabilizing (regulating) mechanisms' (Clark *et al.*, 1967).

6.3 Andrewartha and Birch's view

By contrast, the view that density-dependent processes are '... in general, of minor or secondary importance, and ... play no part in determining the abundance of some species' (Clark *et al.*, 1967) is most commonly attributed to two other Australian ecologists,

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Andrewartha and Birch. Their view is as follows (Andrewartha & Birch, 1954):

> The numbers of animals in a natural population may be limited in three ways: (a) by shortage of material resources, such as food, places in which to make nests, etc.; (b) by inaccessibility of these material resources relative to the animals' capacities for dispersal and searching; and (c) by shortage of time when the rate of increase r is positive. Of these three ways, the first is probably the least, and the last is probably the most important in nature. Concerning (c), the fluctuations in the value of r may be caused by weather, predators, or any other component of environment which influences the rate of increase.

Andrewartha and Birch, therefore '... rejected the traditional subdivision of environment into physical and biotic factors and 'density-dependent' and 'density-independent' factors on the grounds that these were neither a precise nor a useful framework within which to discuss problems of population ecology' (Andrewartha & Birch, 1960). The views of Andrewartha and Birch are probably made more explicit, however, by considering one of their examples; subsequent discussion of this example will then lead on to a crystallization of the current status of the controversy.

6.4 An example: *Thrips imaginis*

Davidson and Andrewartha (1948a, b) studied population changes in a phytophagous insect, the apple-blossom thrips *Thrips imaginis* which lives on roses throughout southern Australia. They obtained estimates of abundance for 81 consecutive months by counting the number of thrips on a sample of 20 roses on each of approximately 20 days each month (Fig. 6.1); and then, for a further 7 years, they obtained similar estimates for spring and early summer only. In addition, they monitored local temperature and rainfall throughout the same period. By the use of a 'multiple regression' analysis (see for instance Poole, 1978), they were able to 'account for' 78% of the variance in the yearly peak of thrip numbers by reference to four climatic factors: (i) the suitability of temperature for development up to 31 August; (ii) the suitability of temperature for development in September and October; (iii) the suitability of temperature for development in August of the previous season; and (iv) the rainfall in September and October. In other words, knowledge of these four factors in any one year would allow the size of the peak in thrips numbers to be estimated with a good degree of statistical accuracy.

Clearly, the weather (as represented by these four factors) plays a central and crucial role in the determi-

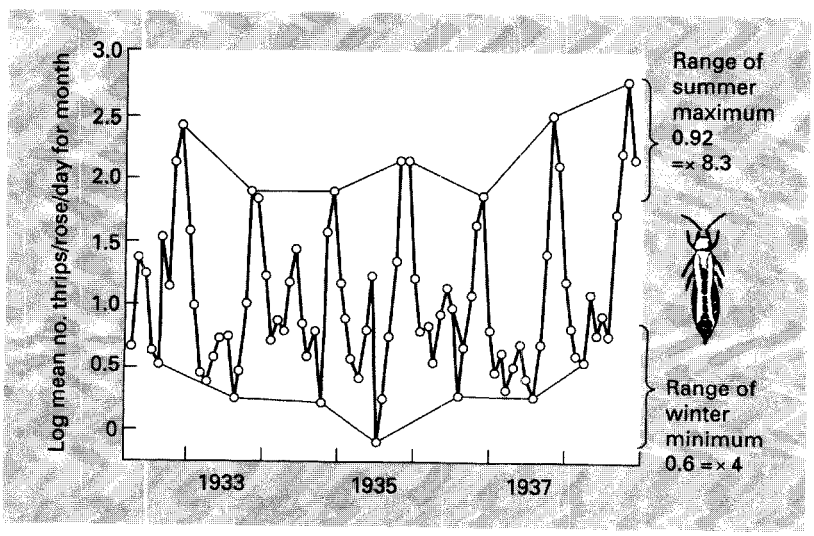


Fig. 6.1 Mean monthly population counts of adult *Thrips imaginis* in roses at Adelaide, Australia (Davidson & Andrewartha, 1948a). (After Varley *et al.*, 1975.)

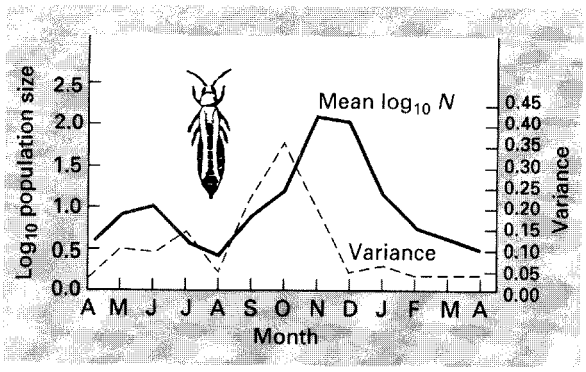


Fig. 6.2 The mean monthly logarithm of population size for *Thrips imaginis* and its variance. (After Smith 1961.)

nation of *T. imaginis* numbers at their peak. Yet Andrewartha and Birch (1954) used this, and the fact that no 'density-dependent factor' had been found, to conclude that there was 'no room' for a density-dependent factor as a determinant of peak thrip numbers. The only 'balance' that Davidson and Andrewartha (1948b) recognized was 'a race against time with the increase in density being carried further in those years when the favourable period lasts longer, but never reaching the point where competition begins to be important'.

As Varley *et al.* (1975) point out, however, the multiple regression model is not designed to reveal directly the presence of a density-dependent factor. And when Smith (1961) applied methods that were so designed, he obtained excellent evidence of density-dependent population growth prior to the peak. He found that:

1 there were significant negative correlations between population *change* and the population size immediately preceding the spring peak; and

2 there was, at the same time, a rapidly decreasing variance in population size (Fig. 6.2).

Moreover, Varley *et al.* (1975) were able to suggest the existence of a strongly density-dependent *mortality* factor, *following* the peak density. They did this by the use of two hypothetical examples (Fig. 6.3). In both cases, the adult population (N_A) is given a reproductive rate of 10, but is then subjected to a random (i.e. density-independent, perhaps climatic) factor determining the (peak) number of larvae (N_L). (Note that this random element, plotted as a *k*-factor, is the same in Fig. 6.3a and 6.3b.) In addition, however, Fig. 6.3a shows a *weakly* density-dependent mortality factor acting on the larvae to determine the next generation's adult population size, while Fig. 6.3b has a *strongly* density-dependent factor. *As a consequence of this*, the climatic factor accounts for 91% of the variation in N_L in Fig. 6.3b, but only 32% in Fig. 6.3a. It appears, in short, as if a fairly strong density-dependent factor must also have been acting on the thrip population between the summer peak and the winter trough; and, indeed, its presence can be

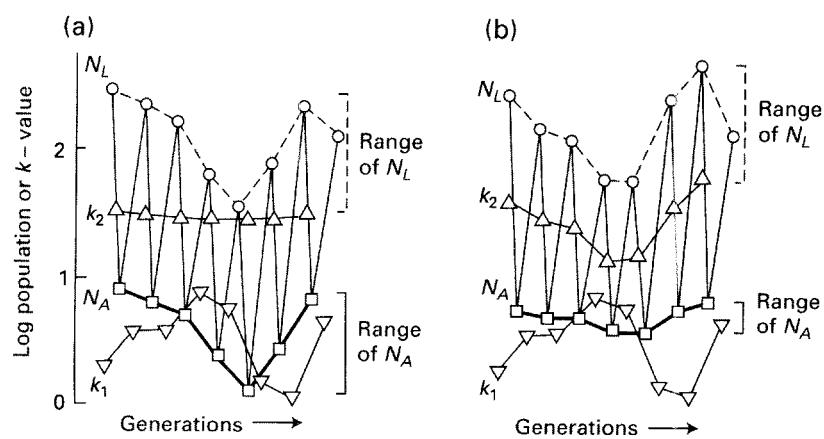


Fig. 6.3 In both (a) and (b) an adult population, N_A, is given a ten-fold reproductive rate. A random factor (represented by k_1) then acts to leave a larval density, N_L, which is plotted on the top line; (a) has a weakly density-dependent factor (k_2); (b) has a strongly density-dependent factor. For further explanation, see text. (After Varley *et al.*, 1975.)

inferred from Fig. 6.1 itself, where the maxima are spread over an eight-fold range, with the minima covering only a four-fold range. In fact, Davidson and Andrewartha themselves (1948b) felt that weather acted as a density-dependent component of the environment during the winter, by killing the proportion of the population inhabiting less favourable 'situations'. (If the number of safe sites is limited and remains roughly constant from year to year, then the number of individuals outside these sites killed by the weather will increase with density.) They also felt, however, that this did not fit the general, density-dependent theory '... since Nicholson (1933, pp. 135–6) clearly excludes climate from the list of possible "density-dependent factors".'

6.5 Some general conclusions

There are several conclusions of general importance that we can draw from this single example:

1 We must begin by distinguishing clearly between the *determination* of abundance and its *regulation*. Regulation has a well-defined meaning (section 2.3), and *by definition* can only occur as a result of a density-dependent process. Conversely, abundance will be *determined* by the combined effects of all the factors and all the processes that impinge on a population. Certain other terms, particularly the *control* of abundance, do not have a single, well-defined meaning. As Varley *et al.* (1975) stress, a reader should pause and consider carefully what kind of meaning is being attached to such terms.

2 We must also recognize that any dispute as to whether climate (or anything else) *can* be a density-dependent factor is essentially beside the point. In Davidson and Andrewartha's thrips, at the appropriate time of the year, weather confined the insects to a limited amount of favourable 'space'. It is irrelevant whether or not we consider weather or space to be the density-dependent factor. What is important is that the two, together, drove a density-dependent *process*, and that the *effects* of weather on the thrips were, in consequence, density-dependent. Weather, like other abiotic factors, can interact with biotic components of the environment so that no single, simple 'density-dependent factor' can be isolated (cf. section 2.3). We must concentrate on the nature of the processes and effects which emanate from such interactions.

3 Andrewartha and Birch's view that density-dependent processes play no part in determining the abundance of some species clearly implies that the populations of such species are not regulated. Yet, as we have seen, even the data they themselves brought forward contradicts this view. Indeed it is logically unreasonable to suppose that any population is *absolutely free* from regulation. Unrestrained population growth is unknown, and unrestrained decline leading to extinction is extremely rare. Moreover, the fluctuations in almost all populations are at least limited enough for us to be able to describe them as 'common', 'rare', and so on. Thus, we can take it that all natural populations are regulated to some extent, and are therefore, to some extent, influenced by density-dependent processes.

4 Furthermore, Andrewartha and Birch's view that density-dependent processes are *generally* of minor or secondary importance, if it is taken to imply that such processes are unimportant, is not justified either. Even in their own example of thrips, density-dependent processes, apart from regulating the population, were also crucially important in *determining* abundance.

5 Conversely, we must remember that the weather accounted for 78% of the variation in peak thrip numbers. Thus, if we wished to predict abundance or decide why, in a particular year, one level of abundance was attained rather than another, then weather would undoubtedly be of major importance. By implication, therefore, the density-dependent processes would be of only secondary importance in this respect (see also section 6.6).

6 Thus, it would be unwise to go along with Nicholson wholeheartedly. Although density-dependent processes are an absolute necessity as a means of regulating populations and are generally by no means unimportant in determining abundance, they may well be of only minor importance when it comes to explaining particular observed population sizes. Moreover, because all environments are variable, the position of any 'balance-point' is continually changing. In spite of the ubiquity of density-dependent, regulating

(i.e. balancing) processes, therefore, there seems little value in a view based on universal balance with rare non-equilibrium interludes. On the contrary, it is likely that *no* natural population is *ever* truly at equilibrium. This, remember, was the essential reason for our pragmatic definition of 'regulation' in section 2.3.

6.6 A life-table analysis of a Colorado beetle population

These conclusions can be illustrated most effectively by analysing some life-table data in a way which has its roots in the work of Morris (1959) and Varley and Gradwell (1960), and is discussed more fully by Varley *et al.* (1975). As will become clear, the analysis allows us to weigh up the role and importance of each of the various mortality factors, and also allows us to distinguish between factors that are important in determining the total mortality-rate, factors that are important in determining *fluctuations* in mortality-rate, and factors that are important in regulating a population. In the present case, the analysis is applied to a population of the Colorado potato beetle (*Leptinotarsa decemlineata*) in eastern Ontario where it has one generation per year (Harcourt, 1971).

6.6.1 Life-table data

'Spring adults' emerge from hibernation around the middle of June, when the potatoes are first breaking through the ground. Within 3–4 days oviposition begins, extending for about 1 month and reaching its peak in early July. The eggs are laid in clusters (average size 34 eggs) on the lower leaf surface and the larvae crawl to the top of the plant where they feed throughout their development passing through four instars. Then, when mature, they drop to the ground and form pupal cells in the soil. The 'summer adults' emerge in early August, feed, and then re-enter the soil at the beginning of September to hibernate and become the next season's 'spring adults'.

Details of the sampling methods can be found in Harcourt (1964), but it should be stressed that:

1 on each occasion, sampling was continued until population estimates had confidence limits which were 10% or less of the mean; and

2 the timing of samples varied from year to year, to ensure that the effects of variable climate on the insects' rate of development were allowed for.

The sampling programme provided estimates for seven age-intervals, from which a life table could be constructed. These were: eggs, early larvae, late larvae, pupal cells, summer adults, hibernating adults and spring adults. In addition, one further category was included, 'females × 2', to take account of any unequal sex ratios amongst the summer adults.

Table 6.1 lists these age-intervals and the numbers within them for a single season, and also gives the major 'mortality factors' to which the deaths between successive intervals can be attributed. Figures obtained directly from sampling are indicated in bold type, the rest were obtained by subtraction. Amongst the eggs, predation and cannibalization were monitored directly, since both processes left behind recognizable egg remains. Conversely, reductions in hatchability due either to infertility or rainfall (mud splash), were assessed by returning samples of eggs to the laboratory and observing their progress individually. Finally, the figure for eggs 'not deposited' was based on the difference between the actual number of eggs and those expected on the basis of spring-adult number and mean fecundity. These five egg 'mortality factors' have, for simplicity, been presented as acting successively in Table 6.1, although, in reality, they overlap considerably. The loss of accuracy resulting from this is generally slight (Varley *et al.*, 1975).

The principal mortality factor during the first larval age-interval (from hatching to second instar) was rainfall, since, during heavy downpours, the small larvae were frequently washed from the leaves to the ground where they died in small puddles of water. This mortality was assessed by taking population counts before and after each period of rain. Conversely, amongst older larvae the major mortality factor was starvation.

Larval mortality due to parasites and predators was insignificant, and well within the sampling error. In the pupal stage, by contrast, parasitization was an important cause of mortality; the numbers of sound

Table 6.1 Typical set of life-table data collected by Harcourt (1971) for the Colorado potato beetle (in this case, for Merivale 1961–62). Figures in bold type were obtained directly, the rest by subtraction.

Age interval	Numbers per 96 potato hills	Numbers 'dying'	'Mortality factor'	$\log_{10} N$	k-value	
Eggs	**11 799**			4.072		
	9268	2531	Not deposited	3.967	0.105	(k_{1a})
	8823	**445**	Infertile	3.946	0.021	(k_{1b})
	8415	**408**	Rainfall	3.925	0.021	(k_{1c})
	7268	**1147**	Cannibalism	3.861	0.064	(k_{1d})
Early larvae	6892	**376**	Predators	3.838	0.024	(k_{1e})
Late larvae	**6892**	**0**	Rainfall	3.838	0	(k_2)
Pupal cells	**3170**	3722	Starvation	3.501	0.337	(k_3)
Summer adults	**3154**	**16**	*D. doryphorae*	3.499	0.002	(k_4)
Female × 2	3280	**– 126**	Sex (52% female)	3.516	– 0.017	(k_5)
Hibernating adults	16	**3264**	Emigration	1.204	2.312	(k_6)
Spring adults	**14**	**2**	Frost	1.146	0.058	(k_7)
					2.926	(k_{total})

pupae and those containing puparia of the parasitic tachinid fly, *Doryphorophaga doryphorae* were estimated directly from the sampling data.

Amongst the summer adults, the principal cause of 'mortality' was emigration provoked by a shortage of food. This was assessed from a series of direct counts during the latter half of August. Damage by frost, the major mortality factor acting on hibernating adults, was assessed by digging up a sample at the end of April. The number of spring adults was estimated by direct sampling in early July. There was no evidence of spring migration, nor of generation-to-generation changes in fecundity. (Where the data provided more than one estimate of the numbers in a particular stage, these were integrated into a single figure.)

As Table 6.1 shows, k-values have been computed for each source of mortality, and their mean values over 10 seasons for a single population are presented in the second column of Table 6.2. These indicate the relative strengths of the various mortality factors as

Table 6.2 Summary of the life-table analysis for Canadian Colorado beetle populations (data from Harcourt, 1971). b and a are, respectively, the slope and intercept of the regression of each k-factor on the logarithm of the numbers preceding its action; r^2 is the coefficient of determination. (See text for further explanation.)

		Mean	Coefficient of regression on k_{total}	b	a	r^2
Eggs not deposited	k_{1a}	0.095	– 0.020	– 0.05	0.27	0.27
Eggs infertile	k_{1b}	0.026	– 0.005	– 0.01	0.07	0.86
Rainfall on eggs	k_{1c}	0.006	0.000	0.00	0.00	0.00
Eggs cannibalized	k_{1d}	0.090	– 0.002	– 0.01	0.12	0.02
Egg predation	k_{1e}	0.036	– 0.011	– 0.03	0.15	0.41
Larvae 1 (rainfall)	k_2	0.091	0.010	0.03	– 0.02	0.05
Larvae 2 (starvation)	k_3	0.185	0.136	0.37	– 1.05	0.66
Pupae (*D. doryphorae*)	k_4	0.033	– 0.029	– 0.11	0.37	0.83
Unequal sex ratio	k_5	– 0.012	0.004	0.01	– 0.04	0.04
Emigration	k_6	1.543	0.906	2.65	– 6.79	0.89
Frost	k_7	0.170	0.010	0.002	0.13	0.02

contributors to the total rate of mortality within a generation. Thus, the emigration of summer adults has by far the greatest proportional effect, while the starvation of older larvae, the frost-induced mortality of hibernating adults, the 'non-deposition' of eggs, the effects of rainfall on young larvae and the cannibalization of eggs all play substantial roles.

6.6.2 'Key-factor' analysis

What the second column of Table 6.2 does not tell us, however, is the relative importance of these factors as determinants of the year-to-year fluctuations in mortality (remember sections 6.4 and 6.5). We can easily imagine, for instance, a factor that repeatedly takes a significant toll from a population, but which, by remaining constant in its effects, plays little part in determining the particular rate of mortality (and thus the particular population size) in any one year. In other words, such a factor may, in a sense, be important in determining population size, but it is certainly not important in determining changes in population size, and it cannot help us understand why the population is of a particular size in a particular year. This *can* be assessed, however, from the third column of Table 6.2, which gives the regression coefficient of each individual k-value on the total generation value, k_{total}. Podoler and Rogers (1975) have pointed out that a mortality factor that is important in determining population change will have a regression coefficient close to unity, because its k-value will tend to fluctuate in line with k_{total} in terms of both size and direction. A mortality factor with a k-value that varies quite randomly with respect to k_{total}, however, will have a regression coefficient close to zero. Moreover, the sum of all the regression coefficients within a generation will always be unity. Their values will, therefore, indicate their relative importance as determinants of fluctuations in mortality, and the largest regression coefficient will be associated with the *key factor causing population change* (Morris, 1959; Varley & Gradwell, 1960).

In the present example, it is clear that the emigration of summer adults, with a regression coefficient of 0.906, is the key factor; and other factors (with the possible exception of larval starvation) have a negligible effect on the changes in generation mortality, even though some have reasonably high mean k-values. (A similar conclusion could be drawn, in a more arbitrary fashion, from a simple examination of the fluctuations in k-values with time (Fig. 6.4). Note, moreover, that Podoler and Roger's method, even though it is less

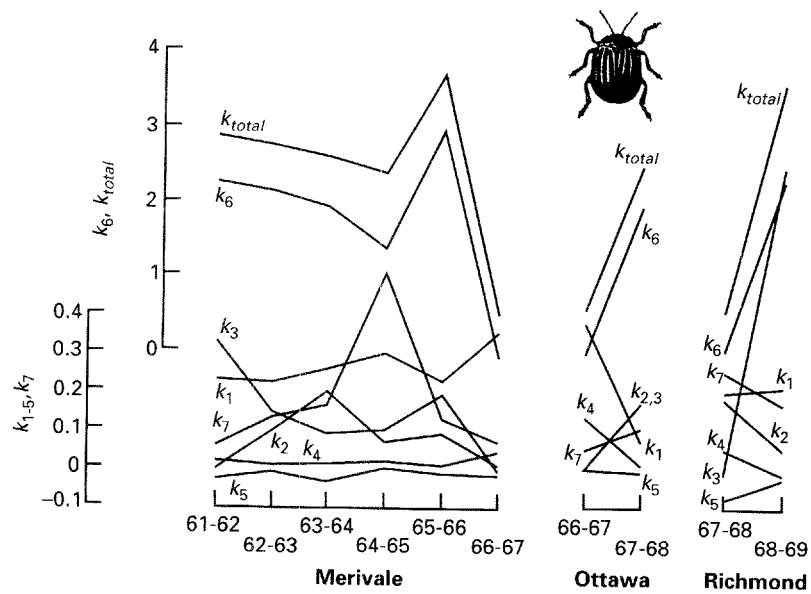


Fig. 6.4 The changes with time of the various k-values of Canadian Colorado beetle populations. Note that there are two quite separate scales on the vertical axis and that k_6 is therefore undoubtedly the 'key factor'. (Data from Harcourt, 1971.)

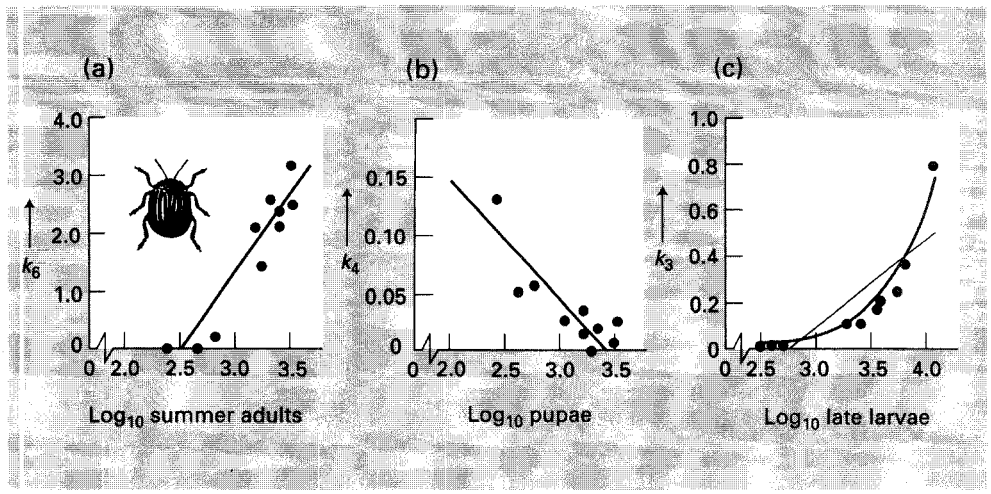


Fig. 6.5 (a) Density-dependent emigration amongst Canadian Colorado beetle summer adults which is overcompensating (slope = 2.65). (b) Inverse density-dependence in the parasitization of Colorado beetle pupae (slope = −0.11). (c) Density-dependence in the starvation of Colorado beetle larvae (straight line slope = 0.37; final slope of curve, based on equation 3.4 = 30.95). (Data from Harcourt, 1971.)

arbitrary than this graphical alternative, still does not allow us to assess the statistical significance of the regression coefficients, because the two variables are not independent of one another.)

Thus, while mean k-values indicate the average strengths of various factors as causes of mortality in each generation, key-factor analysis indicates their relative strengths as causes of yearly changes in generation mortality, and thus measures their importance as determinants of population size.

6.6.3 Regulation of the population

We must now consider the role of these factors in the *regulation* of the Colorado beetle population. In other words, we must examine the density-dependence of each of these factors. This can be achieved most easily by using the method established in Chapter 2, of plotting k-values for each factor against the common logarithm of the numbers present before the factor acted. Thus, columns 4, 5 and 6 in Table 6.2 contain, respectively, the slopes, intercepts and coefficients of determination of the various regressions of k-values on their appropriate '$\log_{10}$ initial densities'. Three factors seem worthy of close examination.

The emigration of summer adults (the key factor) appears to act in an *over*compensating density-dependent fashion, since the slope of the regression (= 2.65) is considerably in excess of unity (Fig. 6.5a). (Once again, there are statistical difficulties in assessing the significance of this regression coefficient, but these can be overcome (Varley & Gradwell, 1963; Varley *et al.*, 1975; Vickery & Nudds, 1991), and in the present case density-dependence *can* be established statistically.) Thus, the key factor though density-dependent does not so much regulate the population as lead, because of overcompensation, to violent fluctuations in abundance (actually discernible from the data: see Fig. 6.7). Indeed, the potato/Colorado beetle system is only maintained in existence by humans, who prevent the extinction of the potato population by replanting (Harcourt, 1971).

The rate of pupal parasitism by *D. doryphorae* (Fig. 6.5b) is apparently inversely density-dependent (though not significantly so, statistically), but because the mortality-rates are small, any destabilizing effects this may have on the population are negligible. Nevertheless, it is interesting to speculate that at low population levels presumably prevalent before the creation of potato monocultures by man, this parasitoid

could act as an important source of beetle mortality (Harcourt, 1971).

Finally, the rate of larval starvation appears to exhibit undercompensating density-dependence (though statistically this is not significant). An examination of Fig. 6.5c, however, indicates that the relationship would be far better reflected, not by a linear regression, but by a curve of the type discussed and examined in seciton 3.2.2. If such a curve is fitted to the data, then the coefficient of determination rises from 0.66 to 0.97, and the slope (b-value) achieved at high densities is 30.95 (though it is, of course, much less than this in the density range observed). Hence, it is quite possible that larval starvation plays an important part in regulating the population, prior to the destabilizing effects of pupal parasitism and adult emigration.

6.6.4 A population model

This type of analysis of life table data allows us to examine the role and important of each of the various mortality factors acting on a population. It also illustrates the differences between factors that are important in determining year-to-year changes in mortality-rate, and factors that are important in regulating (or even destabilizing) a population. The final logical stage in such an analysis is to construct a synthetic model that will allow us to predict:

1 the probable future progress of a given population; and

2 the consequences to the population of natural or enforced changes in any of the mortality factors.

Such a model should have as many 'steps' as there are 'stages' in the original analysis, and for each step there are two forms that the model could take. Ideally, the mortality-rate at a particular stage should be estimated directly from data on the mortality factor itself. Thus, the mortality-rate of early larvae, for instance, should be estimated from rainfall data, and that of pupae from data on the numbers of *D. doryphorae*. Yet, the construction of these specific submodels for each of the stages requires extensive collection of data. An imperfect but simpler alternative, and the one to which we shall be restricted in the present case, is to use the observed, empirical relationships between beetle numbers and mortality-rates, as summarized in the regression equations of columns 4 and 5 of Table 6.2. This approach literally uses what has happened in the past to predict what is likely to happen in the future but it neglects any detailed consideration of the interactions occurring at each of the stages. (Of course, hybrid models, using different approaches at different stages, can also be constructed.)

In the present case we can argue from Table 6.2 that since, on average:

$$k_{1a} = 0.27 - 0.05 \log_{10}(\text{total potential eggs})$$

or,

$$\log_{10}(\text{total potential eggs}) - \log_{10}(\text{eggs deposited}) = 0.27 - 0.05 \log_{10}(\text{total potential eggs})$$

then,

$$\log_{10}(\text{eggs deposited}) = 1.05 \log_{10}(\text{total potential eggs}) - 0.27.$$

Similarly,

$$\log_{10}(\text{fertile eggs}) = 1.01 \log_{10}(\text{eggs deposited}) - 0.07,$$

and so,

$$\log_{10}(\text{fertile eggs}) = 1.01 \{1.05 \log_{10}(\text{total potential eggs}) - 0.27\} - 0.07.$$

If this is repeated for each stage, it allows us, eventually, to predict $\log_{10}$ (spring adults) from $\log_{10}$ (total potential eggs). In other words, we have constructed a model that will allow us to predict the level of infestation in any one year given the number of eggs laid the previous year. Alternatively, since we know that each female lays, on average, 1700 eggs, we can use the number of spring adults in any one year to predict the numbers in the various stages during the following year. In the present case, the model's predictions are illustrated in Fig. 6.6 (in which 'total potential eggs' is used to predict 'spring adults' in each of the 10 generations studied) and Fig. 6.7 (in which the number of eggs present in 1961 at a single site is used to generate an adult population curve for the

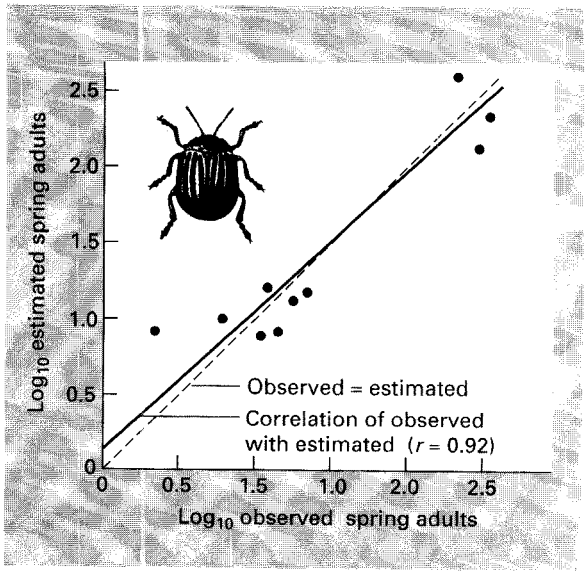


Fig. 6.6 The correlation between the observed number of Canadian Colorado beetle spring adults and the number estimated on the basis of the *k*-value model. For further explanation, see text. (Data from Harcourt, 1971.)

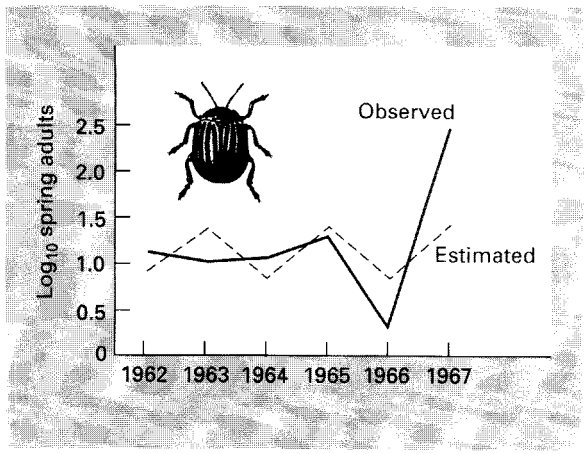


Fig. 6.7 The observed and estimated numbers of Colorado beetle spring adults at Merivale, based on the numbers observed in 1961. For further explanation, see text. (Data from Harcourt, 1971.)

following six seasons). In both cases the predictions are compared with the observed figures, and, given that in Fig. 6.7 any errors are bound to accumulate, the fit of the model is very satisfactory. This is a pleasing reflection of the amount that we can learn from such analyses of life-table data; and it serves to re-emphasize the need to consider carefully the integrated effects of all factors, both biotic and abiotic, when we seek to understand the abundance of natural populations.

6.7 The problem re-emerges

In recent years a density-dependence debate has resurfaced, but is now focused on the detection of density-dependence rather than on the roles of density-dependent and density-independent factors in population regulation. As many populations persist for long periods within some bounds there can be no doubt that they experience some form of density-dependent limitation. However, the difficulty is in trying to establish whether the density-dependent effects are strong enough to outweigh the stochastic, density-independent effects (Vickery & Nudds, 1991). Two main methods have been used for investigating and detecting density-dependence. The first method has been the detection of density-dependence in particular mechanisms, such as specific parasitoids, predators or competitors, from life-table data, the technique described in section 6.6 for the Colorado potato beetle. The second method has had as its objective the detection of density-dependence of population change from census data, counts made in successive annual generations at the same stage in the life cycle. Several statistical techniques have been used in order to achieve this end (Williamson, 1972; Bulmer, 1975; Slade, 1977; Vickery & Nudds, 1984, 1991; Pollard, *et al.*, 1987; Turchin, 1990). These techniques are a substitute for more extensive analyses such as key-factor analysis. Each of these two main methods are now seen to have problems which are considered in sections 6.7.1 and 6.7.2, respectively.

6.7.1 Life-table analyses

Dempster (1983) challenged the view that populations of temperate Lepidoptera were regulated by density-dependent mortalities from a study of 24 sets of

life-table data. By plotting k-values against the logarithm of the population density upon which the mortality operates (see section 6.6), he found no evidence for density-dependence in eight studies, density-dependence due to intraspecific competition in a further 13 studies, and density-dependence due to natural enemies in just three. Dempster even questioned whether the density-dependent mortality from natural enemies was sufficient to regulate the three populations in question. If Dempster's results were representative for reasonably well-studied groups like Lepidoptera, they could also be true for other groups and have wider implications for our views of population regulation in general. The theme introduced by Dempster was carried further by Stiling (1988), who found that in 26 of the 58 insect species studies he reviewed, there were no obvious density-dependent factors operating and that natural enemies were acting in a density-dependent manner in just 13 studies. This led him to conclude, like Dempster, that population densities of insects commonly fluctuate between some lower floor and upper ceiling set by limiting resources, rather than being maintained around some more fixed equilibrium. What these studies illustrate is that either density-dependent factors, particularly involving natural enemies, are not operating as frequently as was earlier supposed in insect populations, or the methods of detecting density-dependence from life-table data are not as sensitive as could be hoped.

One of the principal problems in detecting density-dependence from life-table data is that the methods used, and described in section 6.6, assume that the important mortality rates are functions of *average* population sizes in successive generations. Consequently, these methods are not designed to test for density-related processes that act differentially between different fractions of the population within a generation. This kind of heterogeneity within a generation can occur when there is differential survival between patches of different population density. We have already seen that such heterogeneity can have far-reaching consequences for competitive (section 4.16) and predator–prey (section 5.13) interactions. For example, if parasitoids search non-randomly among patches of different host density, and concentrate their efforts in high host density patches, then individuals within high density patches may be at a greater risk than those in low density patches. Thus, in high density patches there could be density-dependent mortality, and in low density patches inverse density-dependent mortality; patches of intermediate density would show density-independent mortality. The average mortality over all patch types might be density-independent, which is what would be detected by key-factor analysis. The population of the host would nevertheless be regulated due to density-dependent mortality in the high host density patches. Such a phenomenon has been called spatial density-dependence.

The problem of detecting density-dependence from life tables when there are spatial effects can be illustrated by reference to the work of Southwood and Reader (1976) and Hassell *et al.* (1987) on the population dynamics of the viburnum whitefly (*Aleurotracheus jelinekii*). *Viburnum tinus*, an evergreen shrub, is the main British host for *Aleurotracheus jelinekii*. Whiteflies appear at the end of May or early June and show little migratory activity so that the study bush (B in Hassell *et al.*, 1987) is effectively a closed system. The life cycle is as follows. Up to 30 eggs are laid per female in wax-coated clusters on the undersides of leaves. The eggs hatch after about 4 weeks, leaving the egg cases attached to the leaves. The larvae spend their lives, after some wanderings in the first instar, on the lower leaf surfaces. By November most individuals have reached instar four in which they remain until feeding resumes again the following spring. Over 90% of leaves normally survive into a second season. Two sampling methods were used. An annual census was taken prior to adult emergence in spring, from which the data for key-factor analysis were derived. Further data were collected from more detailed studies on labelled leaves of 30 cohorts per generation which were followed from egg to emerging adult. Thus conventional temporal data is provided from the annual censuses and within-generation mortality data are provided from the studies on individual leaves. Key-factor analysis on 16 generations of data fails to detect any evidence of density-dependent mortality.

However, in 8 of the 9 years in which within-leaf studies were made, density-dependent mortality was detected. The failure to detect density-dependence from the conventional life-table data is the direct result of the failure to sample on the scale at which this density-dependence operates, probably in conjunction with the inevitable stochastic elements present.

It is important, then, in undertaking life-table studies, to sample at a range of spatial scales. Heads and Lawton (1983) have done this in their study of the natural enemies of the holly leaf-miner (*Phytomyza ilicis*), an agromyzid fly. They measured leaf-miner densities along a holly hedge, and mortalities imposed by a guild of specific parasitoids and birds, using nested quadrats varying in size from 0.03 to 1 m^2. The mortalities imposed by three species of pupal parasitoid were independent of density at all four spatial scales, but the larval parasitoid, *Chrysocharis gemma*, revealed a range of responses to host density. In areas of high host density it aggregated demonstrably, and this effect was most notable at the lowest quadrat size. As the data were pooled into increasingly large sampling units the effect became progressively weaker, and was undetectable at the largest quadrat size. The only real solution to the problem is to look for more detailed information from each generation, and to design a sampling programme that is stratified in space (Hassell, 1985).

There are two additional very different and very important reasons why analyses of life-table data have failed to detect density-dependent mortalities. First, Turchin (1990) point out that conventional life-table analyses fail to detect *delayed* density-dependence because they are simply not designed to detect it. He evaluated the evidence for density-dependence in the population dynamics of 14 species of forest insects, and assessed the effect of regulation lags on the likelihood of detecting direct density-dependence. In five of these 14 cases there was clear evidence for direct density-dependence, but seven of the nine apparently non-regulated populations, were, in fact, subject to delayed density-dependent regulation. The implication of this work is that populations characterized by delays in regulation brought about by, for example, natural enemies, will be indistinguishable from those apparently displaying no density-dependent effects. Such populations could have been prominent in the studies of Dempster (1983) and Stiling (1988).

Second, many of the data sets analysed by Stiling were not sufficiently long to make the detection of density-dependence very likely. Hassell *et al.* (1989) analysed the same life tables as Stiling and found that the proportion of studies in which density-dependence was detected by the original authors increased markedly with the number of generations available for analysis. The result was particularly clear in 28 life tables from univoltine insects. This is an encouraging result when set against the problems of spatial density-dependence described above.

6.7.2 Single-species time series

Several statistical tests have been devised with which to analyse annual census data. The simplest ones involve analyses of plots of X_{t+1} against X_t, where $X_t = \log_e N_t$ and $X_{t+1} = \log_e N_{t+1}$, N_t being the population size at time t and N_{t+1} population size at time $t+1$ (e.g. Slade, 1977). Other tests are a little more complicated. For example Bulmer's (1975) autoregression technique specifically contrasts the density-independent null hypothesis of population change, $X_{t+1} = X_t + \varepsilon_t$, where ε_t is a normally distributed error term, against the density-dependent alternative hypothesis, $X_{t+1} = \alpha X_t + r + \varepsilon_t$, where α and r are constants. The randomization method of Pollard *et al.* (1987) uses the correlation coefficient between the change in population size and the population size itself to compare the observed data with randomized sets of annual abundances, which define the null hypothesis of density-independence. None of these tests is perfect and there are unresolved problems associated with them. For example, none of the tests takes account of measurement errors, which are thought to lead to an increased likelihood of detecting density-dependence where none exists. Most authors attempting to analyse census data for density-dependence have used several methods and taken a consensus view of the results.

Some analyses of annual census data have clearly

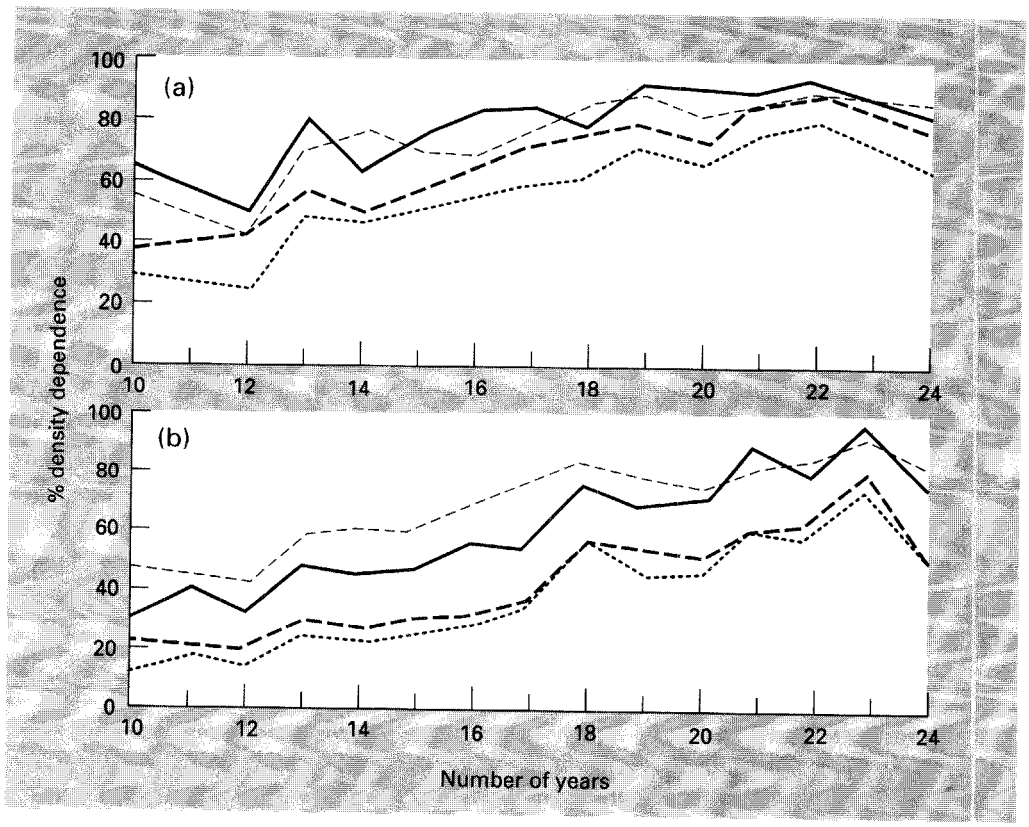


Fig. 6.8 Percentage of time series with significant (5%) density-dependence in (a) aphids, and (b) moths detected using three methods: (——) Bulmer; (– – – –) Ricker; (– – – –) Pollard *et al.*; (-----) all methods. (After Woiwod & Hanski, 1992.)

been deficient on several grounds, most notably, as with the life-table data, on sample size. Many of the statistical tests employed to detect density-dependence have low statistical power with the samples sizes frequently used. This problem does not affect the most extensive study of density-dependence on insect populations by Woiwod and Hanski (1992) which uses the large data base collected by the Rothamsted Insect Survey. This survey includes time-series data for hundreds of species of moths and aphids collected simultaneously from light traps (moths) and suction traps (alate aphids) throughout the UK. In 1976 there were 126 light traps run by 460 volunteers (Taylor, 1986). The data set does not suffer from the same degree of bias as other data in that outbreak species, typically those of greatest interest to applied entomologists, are not disproportionately represented; the species chosen select themselves by getting caught in standard traps. Woiwod and Hanski (1992) analysed 5715 time-series of annual abundances of 447 species of moths and aphids in the UK. This was more than two orders of magnitude greater than the number of time-series analysed in comparable studies. They found that the incidence of significant density-dependence increased with the length of the time series in the range 10–24 years, a similar finding to analyses based on life-table data (Fig. 6.8). Density-dependence was detected more frequently in species which did not show a significant trend in abundance. This was thought not to be a statistical artefact; populations showing a systematic change in size through time may indeed exhibit less density-dependence than those showing no such change.

Woiwod and Hanski used three different techniques to detect density-dependence. In what they considered the most sensitive statistical test, due to Bulmer (1975), on data sets longer than 20 years, density-dependence was detected in 79% of moth and 88% of aphid time-series. The analysis also revealed that noctuid moths and aphids showed highly significant differences between species, indicating that the density-dependence detected was a repeatedly measurable characteristic of species. In other words, density-dependence was consistently detected in some species and not others. There was rather less systematic variation between sites, but not surprisingly species did vary between sites if environmental conditions, to which they may be sensitive, differed greatly. It should be remembered that previous studies had often been concerned with unreplicated time-series from which it was often difficult to know what value should be placed on the results. Finally, Hanski and Woiwod (1991) looked at evidence for delayed density-dependence. Their results support Turchin's (1990) view that delayed density-dependence may be relatively common in forest moths, which often have cyclic or outbreak dynamics (woodland noctuid moths showed more delayed density-dependence than non-woodland noctuids), but in the majority of insects Hanski and Woiwod (1991) found no more delayed density-dependence than would be expected by chance. The good agreement between the methods used by Woiwod and Hanski (those of Ricker, 1954; Bulmer, 1975; Pollard *et al.*, 1987) indicates strongly that this extensive work should finally lay to rest any remaining doubts about the extent of density-dependence in natural populations, of insects, at least.

6.7.3 Population regulation in vertebrates

The preceding sections are dominated by insect examples because there are more data for insect populations than for any other group of animals, and because insect pest problems have driven the need to understand insect population ecology. Table 6.3 gives the numbers of studies reporting different causes of density-dependence in vertebrates. The table is derived from Sinclair (1989) from which the source references can be obtained.

Some patterns are very clear from this table. Large terrestrial mammals seem to be regulated through their food supply, a conclusion based on studies in which food supply either has been measured directly or inferred from its effects on fertility. This may appear a somewhat surprising conclusion given the number of studies that have demonstrated an increase in prey numbers following predator removal, but as Sinclair points out, most studies of this kind merely demonstrate that predators limit prey populations, not that they regulate them. In small mammals the biggest single cause of regulation is density-dependent exclusion of juveniles from breeding colonies, but it is difficult to know how much credence to give to this conclusion since predation is so rarely measured in field studies of small mammal populations. In birds

Table 6.3 Number (%) of reports of separate populations recording cause of density-dependence. (After Sinclair, 1989.)

Group	Space *N* (%)	Food *N* (%)	Predators *N* (%)	Parasites *N* (%)	Disease *N* (%)	Social *N* (%)	Total no. populations
Large marine mammals	0	6 (60)	4 (40)	0	0	0	10
Large terrestrial mammals	1 (1)	71 (99)	0	0	2 (3)	0	72
Small mammals	14 (67)	5 (24)	4 (19)	0	0	14 (67)	21
Birds	5 (33)	8 (53)	0	1 (6)	0	7 (47)	15

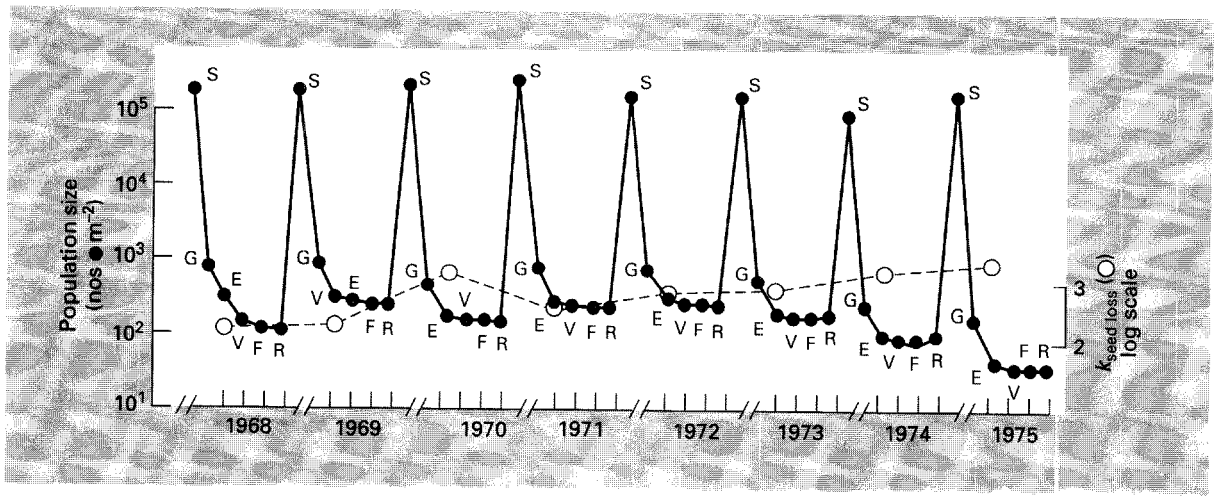


Fig. 6.9 Population fluctuations in a sand dune annual *Androsace septentrionalis:* S, seed population; G, seeding; E, established plant; V, vegetative mature plants; F, flowering plants; R, fruiting plants. Breaks in horizontal axis represent the period from May to March (10 months). (Data from Symonides, 1979.)

food shortage and competition for territorial space and/or nest sites are the main causes when density-dependence is recorded (see also section 6.10). Marine mammals were included in the table by Sinclair (1989) who assumed that food supplies affecting fertility and the growth of juveniles were the predominant causes of regulation. However, the number of studies is so small that no clear conclusions can be drawn. Even less data are available for fish.

It is clear that there are no generalizations about the causes of density-dependent mortality in vertebrates. One of the main reasons for uncertainty is that most studies have not included all the likely contenders. In particular, predation is not considered in most studies and the effects of parasites and pathogens have hardly begun to be considered in field studies.

6.8 Population regulation in plants

Generally two approaches have been taken to examining regulation in natural plant populations. On the one hand, populations fluctuating in size have been monitored by regular census over long periods of time; and on the other hand, deliberate alterations in population size have been made to enable population response to density to be assessed.

Symonides (1979) monitored *Androsace septentrionalis* in sand dunes in Poland over an 8-year period and examined the fluctuations in the numbers of individuals throughuot the life cycle (Fig. 6.9). The life cycle in *Androsace* is short; seedlings emerge in early March and plants have flowered and set seed by late May. Annually, and with considerable consistency during the period 1968–74, over 100 000 seeds were produced per m^2. Yet at fruit dispersal in May of each year, populations fell in the restricted range of 100–300 flowering plants per m^2. Key-factor analysis (see Silvertown, 1982 for details) revealed that the 'key' cause of mortality was seed loss in the 10 months intervening between seed shed in May and germination in the following March. The magnitude of this loss, however, was independent of seed density. On average only 0.4% of the seed crop was present as seedlings in early spring and whilst the exact causes of mortality are not known, this is a clear reflection of the hostility of the sand dune environment for dormant *Androsace* seed. The only other significant period of death during the life cycle was in the establishment of young plants from seedlings. Here density-dependence regulated the number of young plants in an undercompensatory fashion (Fig. 6.10). Such

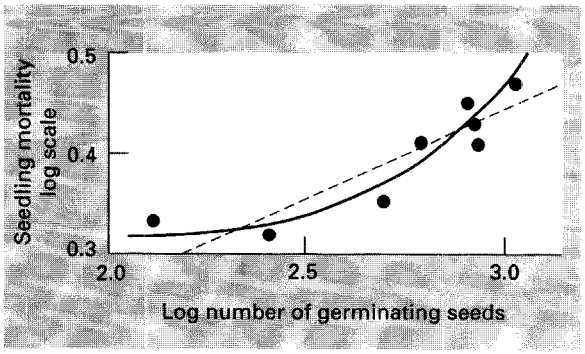


Fig. 6.10 Effect of increasing density on seedling mortality. Slope of dashed line = 0.2 (r^2 = 0.58). Fitting equation 3.4. *b* (final slope) = 18.5 (r^2 = 0.81). (Data from Symonides, 1979.)

undercompensation is important to the persistence of *Androsace* in sand dunes. We can see from Fig. 6.9 that during the latter 5 years of Symonides' study, mortality of dormant seeds from primarily abiotic causes increased ($k_{seed\ loss}$ rises), and in 1975, 98% of seeds were lost after dispersal and the population had declined to 36 plants per m^2. Had density-dependence at the seedling stage been stronger (e.g. exact compensation) it is possible that local populations would have been very close to extinction.

Regulation in this species, then, is substantial in the juvenile stages (seed and seedling), although we might have expected regulation of seed number at the flowering stage as well. *k*-Value analysis of the data revealed no evidence of this but only because the densities of flowering plants had already been restricted. This illustrates the inherent limitation of simply taking population estimates by census and seeking (albeit with a powerful tool) the causes of population regulation. If, for some reason, the range of densities of flowering plants had been greater, density-dependent regulation of seed fecundity may well have been detected.

Sand dune environments offer a particularly hostile habitat for plant growth not least with respect to water as is illustrated particularly clearly by another sand dune annual *Erophila verna*, also studied by Symonides (1983). Over the period 1968–72, mean spring seedling abundance varied no more than 8% of the mean over the 5 years (Fig. 6.11a). During this period, variation in rainfall frequency (as measured by days of recorded rainfall) was slight and, as Figs 6.11b–d show, population regulation occurred both by increased mortality and reduced fecundity in relation to density. The pattern of adult plant survivorship to flowering was found to be strongly dependent on seedling density (the pattern of survivorship changing from Deevey type 1 through to type 3 with increasing density) and the mean fecundity of individual plants was also dependent on the density of survivors. The next 2 years provided very differing habitat conditions with respect to water, 1973 being wetter and 1974 dryer than average. This resource fluctuation was directly reflected in altered peak seedling populations, survivorship to flowering and mean plant fecundity but even so the same processes of density-dependent regulation were evident.

Both of these examples come from studies of natural populations by continued census over long periods of time. An alternative approach to exposing regulatory processes is to alter experimentally the density of natural plant populations. In an endeavour to expose all possible sources of density-dependent regulation in the grass *Vulpia fasiculata* in sand dunes, Watkinson and Harper (1978) studied natural populations in which they deliberately established (by addition of seeds or removal of very young seedlings) a range of densities from 100 to 8000 plants per 0.25 m^2. This species contrasts with *Androsace* in having a shorter period between seed dissemination and germination; seeds 'over-summer' from July through to October, a period in which there is usually less than 1% loss of the annual seed crop. Mortality between seedling emergence and flowering varied between 7 and 41% but was not density-dependent. A range of abiotic and biotic factors were the cause of these deaths, including rabbit grazing, drought and seedling removal by wind drag. This mortality was visited on populations up to and including early flowering. *Vulpia* plants may bear up to four seeds, but as Fig. 6.12a shows, the actual numbers of seeds borne per plant was dependent on the density of plants at flowering time. (Note, though, that below 100 plants per 0.25 m^2, seed number is independent of density.) To examine the stabilizing properties of this density-dependence, Watkinson and

Harper proposed a model in which the density of flowering plants, N_t, was a function of the number of seeds per unit area produced in the previous generation, S_{t-1} and the probability, p, of an individual surviving from seed production (at time $t-1$) through to maturity (at time t). Thus,

$$N_t = pS_{t-1}. \tag{6.1}$$

But, as Fig. 6.12a illustrates, the average number of seeds borne per plant, s, was linearly related to flowering plant density N (above 100 plants per 0.25 m^2). Hence,

$$s = K - C \log N$$

(K and C are the constants describing the straight line relationship) and the seed yield per unit area of these N plants is

$$sN = (K - C \log N)N.$$

Substituting this term with the appropriate subscripts into equation 6.1 gives

$$N_t = p(K - C \log N_{t-1})N_{t-1}$$

a model describing the changes in the number of flowering plants from generation to generation.

If density-independent mortality is constant from one generation to another we may calculate an equilibrium population density ($\hat{N} = N_t = N_{t-1}$; $N_t / N_{t-1} = 1$) as

$$\log \hat{N} = \frac{K}{C} - \frac{1}{Cp}$$

when populations exceed 100 flowering plants per 0.25 m^2. (Below this density, there is no density-dependent regulation and hence no equilibrium population size.) Figure 6.12b shows the predicted equilibrium density for a range of p values and we can see that it becomes increasingly sensitive to lowered chances of survival. When p falls below 0.31, the population will decline; this is because the chance of an individual *Vulpia* plant replacing itself in the next generation is less than 1. Pleasingly, the values of p measured for *Vulpia* in the dunes fell in the range 0.34–0.59 supporting the prediction of the model that populations would persist in the dunes, a fact historically recorded since the beginning of the nineteenth century (Watkinson & Harper, 1978).

The time at which density-independent thinning takes place in the growth of a population of plants may well be important: early thinning may offer a greater period of time than late thinning for the increased growth of survivors compensating for density reductions. The extent of compensation, however, will depend on the time when the various components of yield are formed (section 2.5.2). Watkinson (1983) assessed the importance of this by varying the time during the life cycle when populations were thinned by a half (a figure chosen arbitrarily). Figure 6.12c gives a summary of these effects on the calculated equilibrium population densities. Thinning during the seed phase of the life cycle has no influence on equilibrium density, since surviving plants compensate by elevated seed production (Fig. 6.12a). However, as the time of thinning was successively delayed (dotted line, Fig. 6.12c), equilibrium population densities fell as the compensatory response became increasingly muted. The individual yield components in *Vulpia*—number of fertile tillers, number of spikelets per flower and number of seeds per spikelet—were determined in accordance with the density perceived at their formation, and in consequence compensatory responses to density reductions were limited by the yield components already formed. However, the extent to which this occurred was dependent on the extent to which competition had occurred prior to the time of thinning. Plants sown at a later date (November, Fig. 6.12c) had not achieved a sufficient size to interfere with each other's growth by January and February, and thinning of populations at this time depressed population equilibria to a lesser extent than in populations sown 1 month earlier. We can appreciate, then, that the model developed earlier has the generality to encompass all situations where population regulation is achieved; yet it subsumes within it a density-dependent term which itself is determined by the level of density-independent regulation.

This approach to assessing possible population equilibria may be used in exploring the reasons for the abundance of a species along an environmental gradient. Keddy (1981) sowed seeds of the annual sea

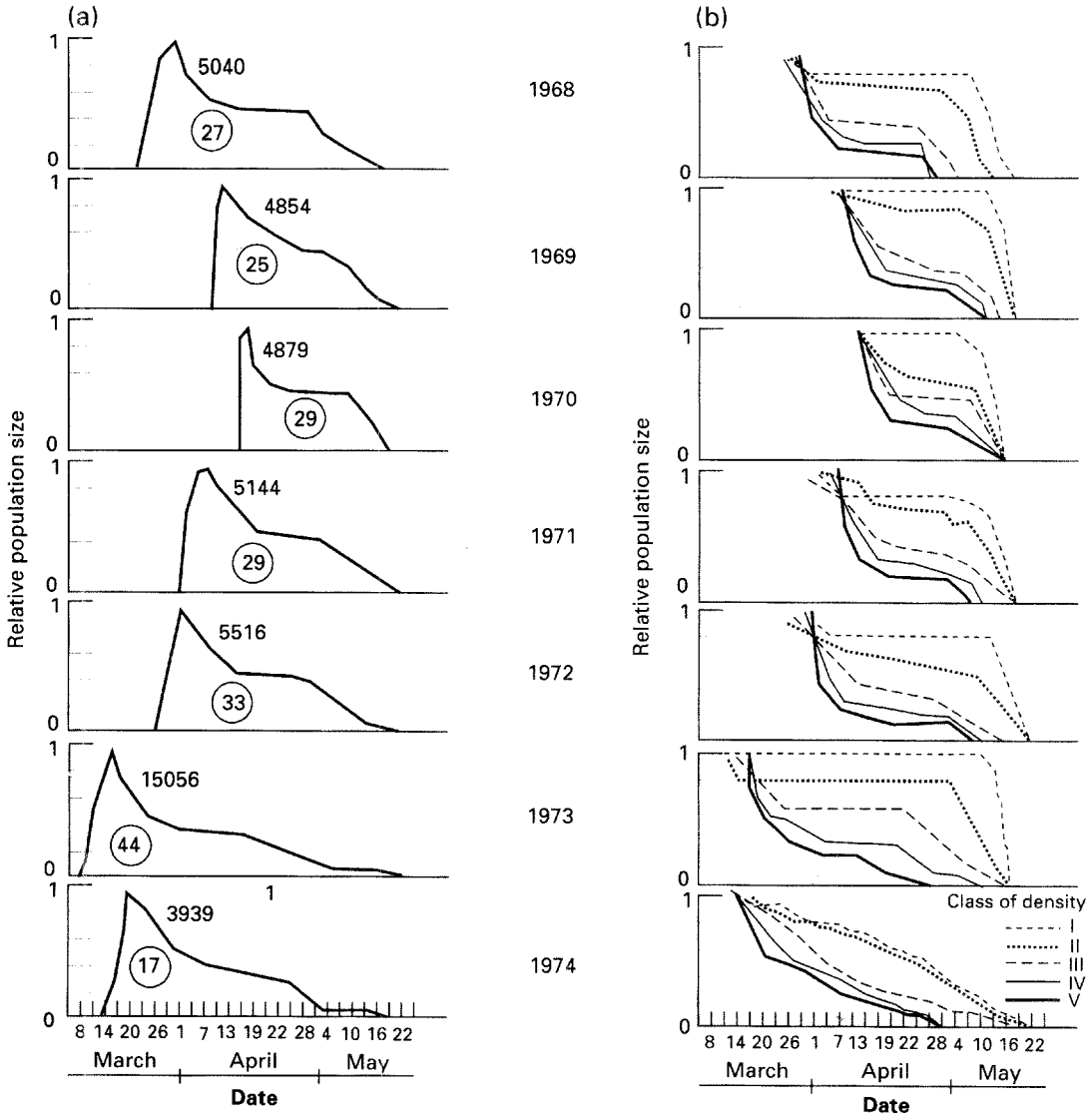


Fig. 6.11 Population responses in *Erophila verna* over a 7-year period. (a) Peak population sizes given are seedlings m^{-2} and the number of days of rainfall in the growing season are circled; (b) survivorship curves for successive cohorts in each year.

rocket (*Cakile edentula*) into a sand dune at three sites (seaward, middle and landward) in Nova Scotia, Canada. A range of densities were sown at each site and plant mortality and fecundity were determined. Density-dependence was only noticeable in two sites and in each, at different stages in the life cycle (Fig. 6.13a). In the landward site survivorship of plants to fruiting was density-dependent whereas in the seaward location fecundity decreased with density. Characteristically, plants were large in the seaward site where populations were largely monospecific in contrast to the much smaller plants at the landward edge of the dune in the presence of other vegetation. Watkinson (1985) used the approach described in

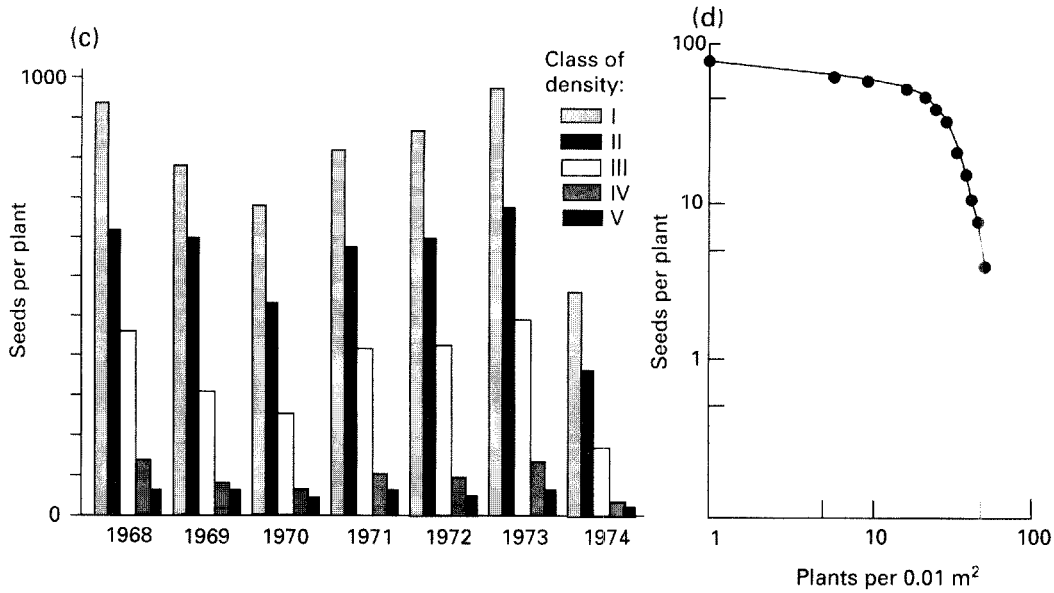


Fig. 6.11 (*continued*) (c) Seed production per plant according to density class; (d) mean plant fecundity in relation to density averaged over the 7 years. Density classes (numbers 0.01 m^{-2}): I, 1–2; II, 5–10; III, 15–30; IV, 35–50, V, > 55. (From Symonides, 1983.)

section 3.2.3 to predict equilibrium population sizes that might be expected if density responses remained constant over generations. Parameter estimates were obtained by fitting equation 3.7 to the observed data (Fig. 6.13b), assuming that self-thinning in populations was largely negligible, that plant mortality was density-independent (the inclusion of density-dependence for the landward site did not qualitatively alter the conclusions) and that 60% of the seed crop in each site were lost in the period after dispersal and before seed germination. By iterating the model for several generations it was found that populations would only persist on the seaward end of the gradient. This is in direct contradiction to the observed relative abundance of sea rocket which was most abundant in the middle of the gradient. The suggests that relative abundance cannot be explained simply in terms of survival and fecundity, assuming of course that the assumptions of the model are correct. Watkinson therefore investigated the influence of migration of seed in the landward direction which is likely as a result of both wind and wave action (Keddy, 1981). It was found that allowing 10% of surviving seeds at the seaward site to migrate inland was sufficient to maintain populations at all three sites and migration levels of 50–80% produced a population distribution with highest abundance in the middle of the dune. The suggestion is that plants exist at the middle and landward sites only because of the high annual dispersal of seeds inland.

In the species just discussed generations do not overlap, either as living plants or as seeds (there is no persistent seed bank) and individuals in the population enter each stage of growth largely synchronously owing to birth (germination) at very much the same time. However, this life history is but one—and perhaps the simplest—that plant species display. Many species possess seed banks from which seedlings may be recruited over a protracted period of time. Episodic germination from a bank of seeds will result in an age-structured population which, as we have already seen (Chapter 3), requires a more sophisticated mathematical description. Yet, despite this complexity we can still unravel sources of population regulation in some instances by straightforward methods.

Figure 6.14 illustrates the density relationships

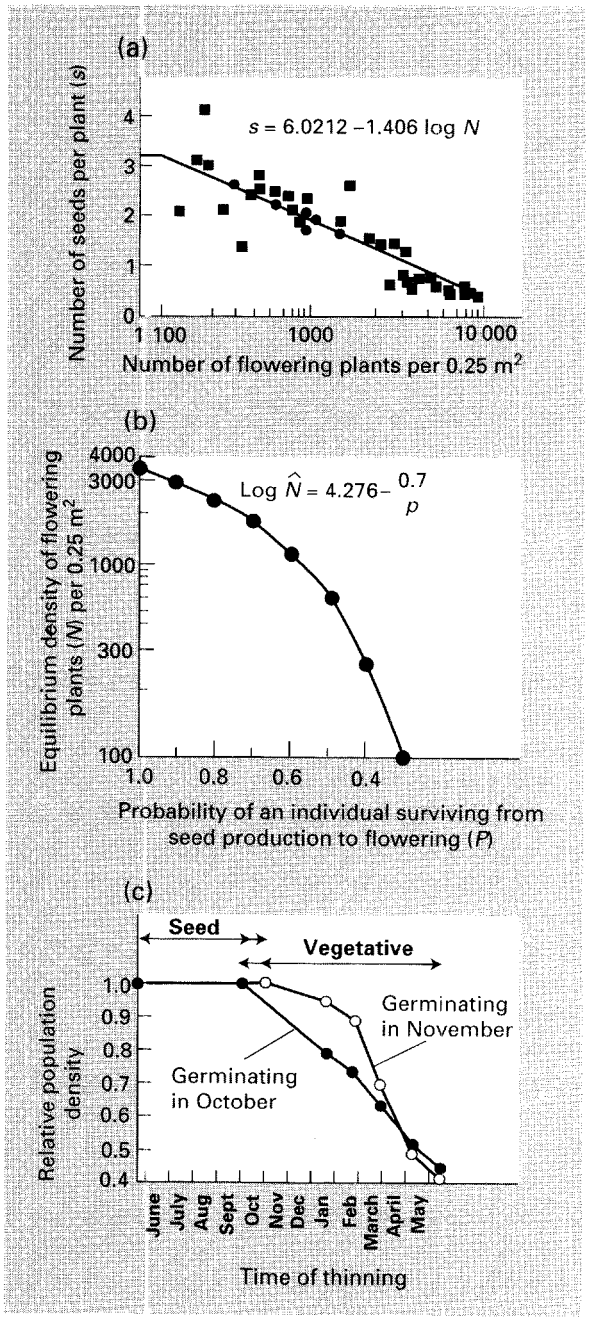
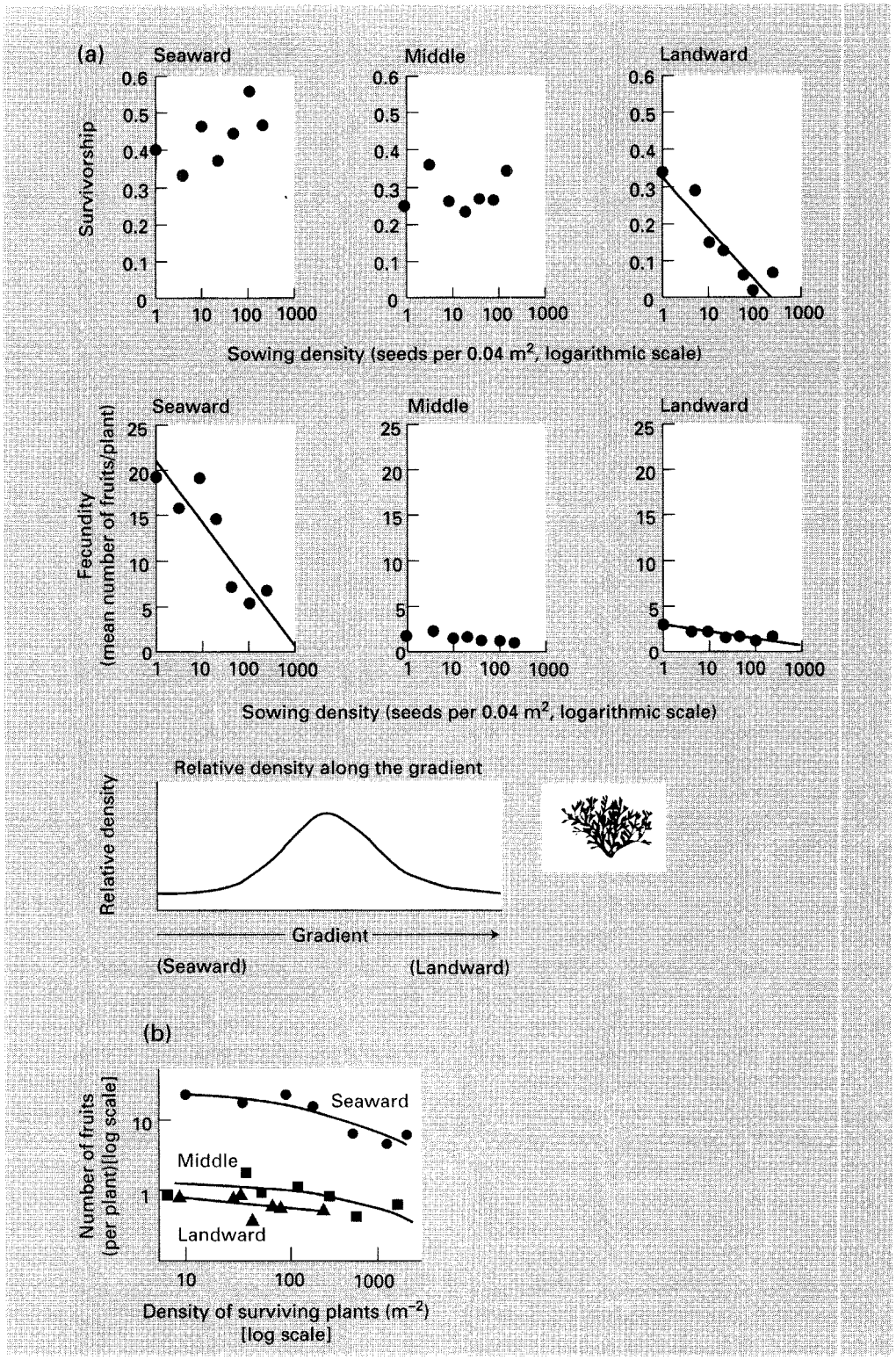


Fig. 6.12 Population regulation in *Vulpia fasiculata*. See text for details. (After Watkinson and Harper, 1978; Watkinson, 1983.)

occurring within populations of the grass weed *Avena fatua* (wild oat) infesting a crop of wheat. In the UK, seed germination in this weed occurs in autumn during crop sowing as well as in spring when the crop is growing. Populations then become age-structured according to the range of seed germination times. Mature plants disseminate seed in mid-summer and die before crop harvest. Manlove (1985) sowed *A. fatua* over a wide range of seed densities on its own or in the presence of a constant density of wheat plants. He then tagged the wild oat plants as they emerged and followed their survivorship through to seed production. By plotting the recorded densities of seeds versus seedlings and seedlings versus adults we can look for regulation at these two stages in the life cycle (Fig. 6.14a & 6.14b). Density-dependent regulation is suggested at the seed/seedling stage: the scatter of points in Fig. 6.14a is below the line of unit slope. Once seedlings became established mortality during growth to plant maturity was slight and not related to seedling density either in the presence or absence of the crop (Fig. 6.14b). Further experimental evidence showed that regulation of seedling number was primarily the result of seed loss from the soil surface in late summer and early autumn, when seed predators (birds and small mammals) foraged in the plant stubbles after harvest. The intensity of this predation increased in a density-dependent manner.

A second source of density-dependent regulation in wild oat was in seed production per plant (Fig. 6.14c). This was inversely related to mature plant density, and it was also depressed eight-fold on average by wheat, across the entire density range. We can examine the stabilizing properties of these two regulatory phases by calculating the net reproductive rate, R, of populations at different starting seed densities

Fig. 6.13 (*Facing page*) The influence of sowing density on fitness in *Cakile edentula* on a sea shore in Nova Scotia, Canada. (a) Fitness components—the proportion of the population surviving to reproduce; the mean fecundity of a surviving plant; and the relative abundance of plants as observed on the sea shore. (After Keddy, 1981.) (b) The relationship between mean fecundity of a plant and the density of reproducing plants. Lines have been fitted using equation 3.7. (After Watkinson, 1985.)


(a)
Seaward
Middle
Landward
Survivorship
0.6
0.5
0.4
0.3
0.2
0.1
0
1
10
100
1000
Sowing density (seeds per 0.04 m², logarithmic scale)
Fecundity (mean number of fruits/plant)
25
20
15
10
5
Relative density along the gradient
Relative density
Gradient
(Seaward)
(Landward)
(b)
Number of fruits (per plant) [log scale]
Seaward
Middle
Landward
Density of surviving plants (m⁻²) [log scale]

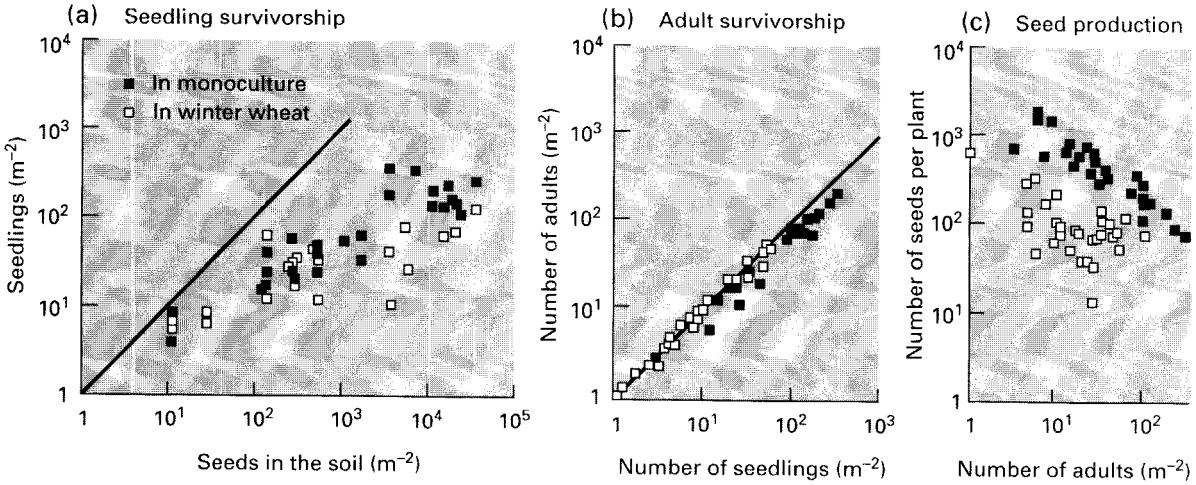


Fig. 6 14 Population regulation in *Avena fatua* in monoculture and in a crop of wheat. (a) Density-dependent regulation of seedling emergence. (b) Density-independent survivorship of adult plants. (c) Density-dependent regulation of seed production. Solid lines are of unit slope. (Data from Manlove, 1985.)

(Fig. 6.15). R Declines monotonically with density in monoculture and in wheat (square symbols) down to densities of *c.* 34 600 and 7240 seeds per m^2 respectively. These are equilibrium population densities—so long as all other factors remain constant from one generation to the next. Figure 6.15 also demonstrates the effect of a density-independent control measure—a herbicide selective against wild oat which was applied in early summer before flowering. Its mode of action is to cause flower abortion in the weed (and hence seed loss) rather than plant mortality. In consequence, reproductive rates are depressed, the line moves towards the origin, and the combination of crop competition and herbicide reduced the equilibrium population density of wild oat to 470 seed per m^2.

This example, whilst supportive of what we already know regarding population regulation, illustrates particularly clearly the various roles of different components of regulation. The underlying cause of regulation is intraspecific. This arises in part from density-dependent seed predation and in part from intraspecific competition determining the seed yield of mature plants. Interspecific competition (from wheat) serves to reduce the net reproductive rate uniformly across the wild oat density range; and a density-independent control (herbicide) depresses these rates even further. We must also remember that the analysis subsumes the effect of age-structure in the wild oat population. Whilst wild oats that emerge in the autumn contribute a greater number of seeds per plant to the next generation than late emergers, these differences are absorbed within the overall density response. Late emerging plants enter the hierarchy of exploitation (section 2.5.2) later in the growing season and are proportionately disadvantaged for doing so. Nevertheless, even individuals lowest in the size hierarchy usually contributed one or two seeds to the next generation.

Part of this study also involved an examination of the dynamics of buried seed populations. Loss of seeds in the soil (through seedling germination and death) occurred at a constant rate (a type 2 survivorship curve) regardless of density. Slightly more than half (0.55) of the seed population in the soil survived from one generation to the next. We can easily appreciate the role of the seed bank in maintenance of plant populations by reconsidering the wild oats growing in wheat and sprayed with herbicide. Populations arising from seed densities greater than the 470 seeds per m^2 had net reproductive rates less than 1—competition and herbicide generally rendering plants at these densities barren. Yet the persistence of dormant seeds

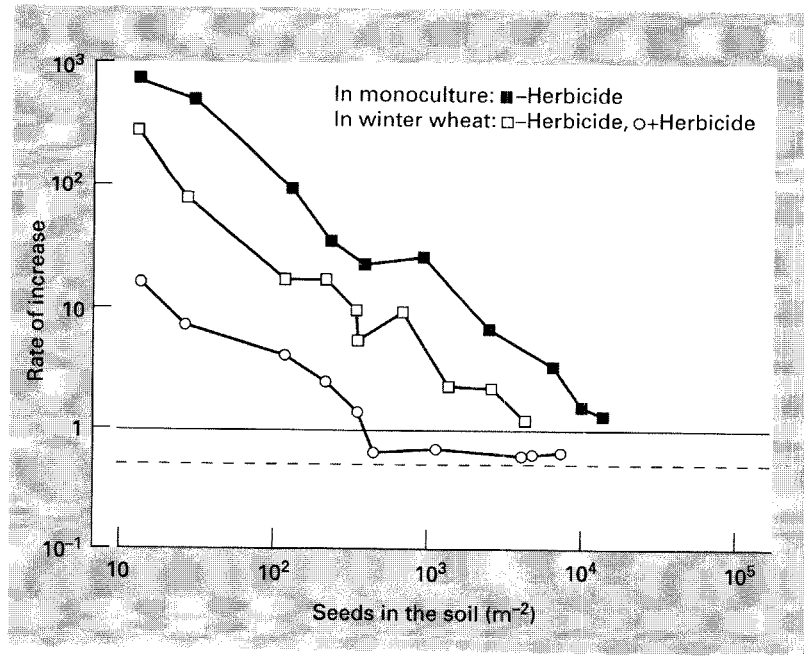


Fig. 6.15 Density-dependent and density-independent regulation in *Avena fatua*. In a generation the number of seeds produced (S_t) is the arithmetic product of: the density of seeds in the soil in the autumn S_{t-1}; the probability of seeding emergence (see Fig. 6.14a); the probability of seedling survival (Fig. 6.14b); and the number of seeds produced per plant (Fig. 6.14c). The rate of increase or net reproductive-rate is then $S_t/S_{t-1} + 0.55$. 0.55 is added as this is the fraction of dormant seeds surviving over generations. The dashed line indicates the (constant) rate of decline of the buried seed population; the solid line shows the rate of increase required to keep a population at equilibrium. The slopes of the lines are not significantly different from one another. (Data from Manlove, 1985.)

over generations enables recruitment of individuals in the succeeding generation and a rapid return to the equilibrium density.

Identifying the precise causes of population regulation in species with a *clonal* growth form is complicated by the necessity of taking into account vegetative propagation by ramet fragmentation (as well as the practical problem of identifying ramets). Two species in which vegetative propagation is common are the creeping buttercup *Ranunculus repens* and the grass *Holcus lanatus*, both occurring in grasslands in the UK. In the buttercup, daughter rosettes are produced on stolons 10–12 cm apart, and in late summer (July–August) these become detached from the parent as interconnecting stolons decay. In *Holcus*, tillers are borne on shoot complexes (< 5 cm apart) which continually become fragmented through natural decay and trampling of grazing animals. In grassland, establishment of new plants from seed in both species is rare. Yet as Fig. 6.16 shows, natural populations of both species undergo considerable turnover whilst maintaining relatively static population sizes. Ramet populations (tillers or rosettes) experienced a constant death risk (type 2 survivorship curve), the life expectancies of ramets in both species being 11–18 months (Sarukhan & Harper, 1973; Weir, 1985). At the same time there was recruitment of new ramets, the process occurring more or less continuously in *Holcus* but with noticeable gain and loss dominated periods in *Ranunculus*. Sarukhan and Harper were able to detect density-dependent regulation in the buttercups as populations accommodated to the pulse of recruitment (Fig. 6.17) whilst in *Holcus* this process was absent because of the fine scale periodicity in ramet

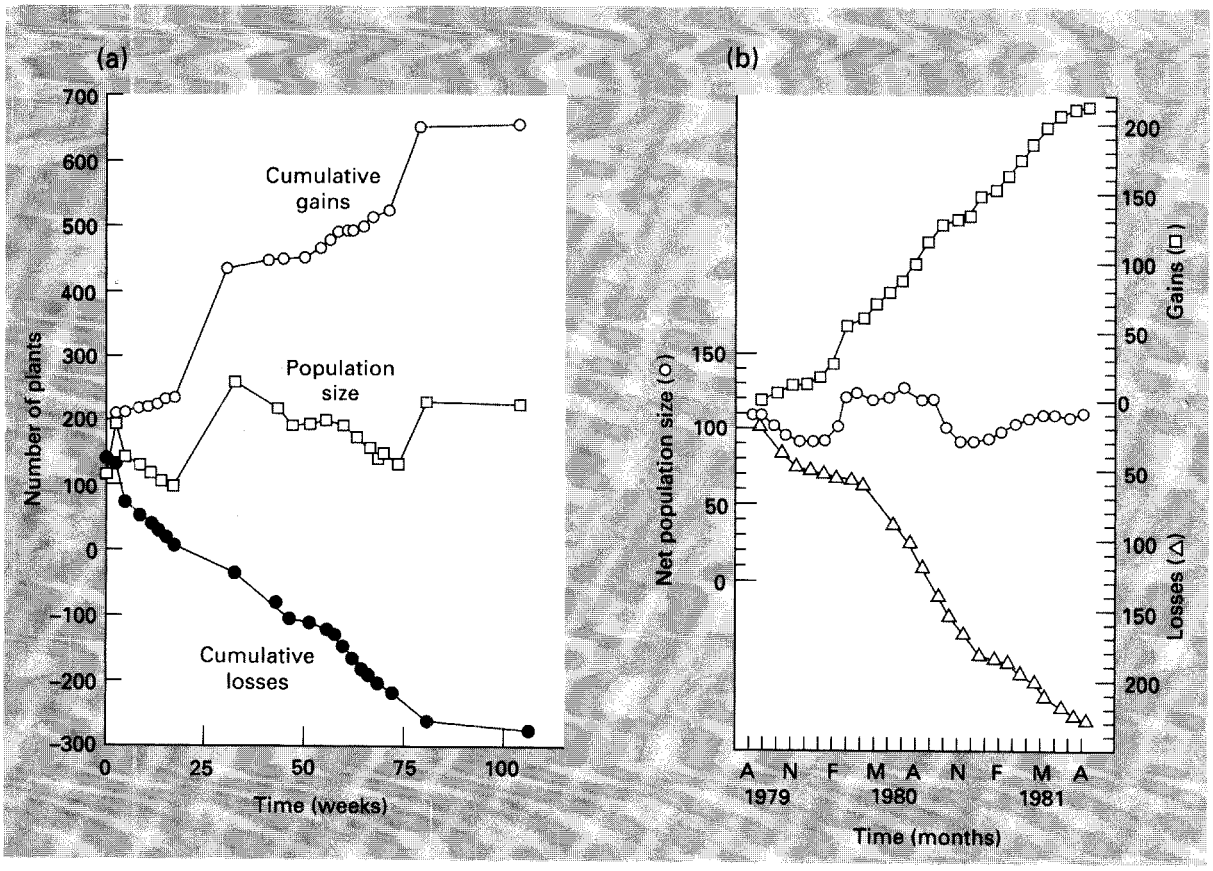


Fig. 6.16 Population turnover in (a) *Ranunculus repens*, and (b) *Holcus lanatus*. (After Sarukhan & Harper, 1973; Weir, 1985.)

replacement. This comparison leads us to a final important conclusion on population regulation in plants. In species where there are pulses of recruitment (as exemplified by unitary species and some clonal ones) there is the opportunity for density-dependent regulation to occur. Conversely, where there is very rapid turnover at the modular level there may be little opportunity for such regulation because of inherent morphological and growth constraints within the growth form. In this case density-dependent regulation may be most likely at the genet level during seedling establishment and occupation of the regeneration niche (section 4.12). It would be unwise to assume that fragmentation is a ubiquitous phenomenon amongst clonal plants. Others may simply increase in size with age and retain structural and physiological integrity. Certainly it has been shown that in some species clones are of considerable age and that they occupy large areas. For instance, Harberd (1961) found clones of a grass *Festuca ovina* in Scottish pastures up to 10 m in diameter and Oinonen (1967) has dated clones of bracken, *Pteridium aquilinum* over 450 years old extending in size over 100 m. The extent to which these clones are intact plants or populations of ramets remains an open question.

6.9 Genetic change

Discussion of population regulation is usually framed in terms of ecological time scales; and although it is generally accepted that the individuals concerned are the products of natural selection genetic change

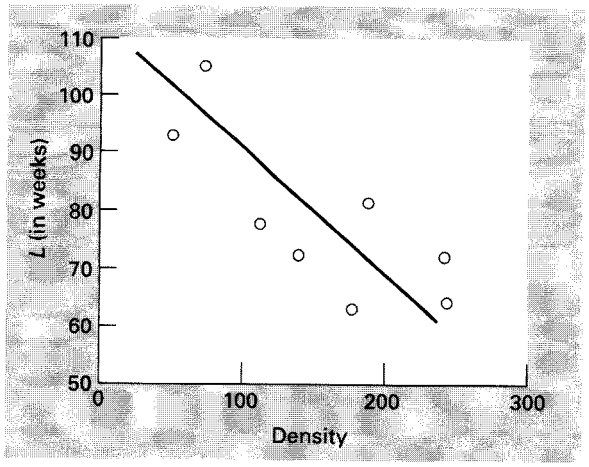


Fig. 6.17 Life expectancy (L) in weeks of vegetative propagules of *Ranunculus repens* as affected by its own species' density. Densities are average values for number of plants (m^{-2}) observed at each site in April 1969, 1970 and 1971. (After Sarukhan & Harper, 1973.)

occurring, by definition, on an evolutionary time scale, is usually neglected as a regulatory mechanism. There are some dissenting ecologists, however—and Pimentel (1961) is the most frequently quoted of these who would suggest that such neglect is unwarranted, and that any discussion of population regulation is incomplete if it fails to take account of contemporary adaptive genetic change. There is, however, rather little concrete evidence in favour of this notion, and it is probable that the ecological and evolutionary time scales are usually dissimilar. Nevertheless, there is some supporting evidence, and a particularly impressive example is provided by the work of Shorrocks (1970). Shorrocks maintained populations of the fruit-fly *Drosophila melanogaster* in the laboratory, and obtained regular four-generation cycles in abundance, unrelated to any regular change in an environmental variable. Pairs of flies were removed from the populations and classified as 'peak' or 'non-peak', depending on the type of abundance their parents experienced, and the numbers of offspring produced by these pairs when maintained under identical, uncrowded conditions were noted. The pairs in the 'peak' category produced significantly fewer offspring than their counterparts in the 'non-peak' category, and, more important, the difference was inherited and remained until the F_1 and F_2 generations. Clearly, demographic characters can respond to selection on an 'ecological' time-scale. In this case they did so in a way that made reproductive-rate inversely related to population size, and thus tended to regulate the population.

6.10 Territoriality

One topic which is intimately tied up with population regulation is territoriality. But as Davies (1978a) has pointed out, in a much fuller review of the subject than can be given here, there are a number of questions pertinent to territoriality which must be kept quite distinct. We can distinguish initially between 'What causes territorial behaviour?' and 'What are the consequences of territoriality?'; but even the first of these is itself the confusion of two quite separate questions, namely 'What is the ultimate cause or driving-force, i.e. what is the selective advantage associated with territoriality?' and 'What is the proximate cause or mechanism through which territories are established?' We shall restrict ourselves here to considering the consequences of territoriality, and the selective advantages associated with it. But first we must define what is meant by a territory and by territorial behaviour. Following Davies (1978a), we will recognize a territory '... whenever [individuals] or groups are spaced out more than would be expected from a random occupation of suitable habitats'. Note, therefore, that territoriality can be ascribed not only to conventional cases like breeding great tits (Fig. 6.18a), but also to barnacles (Fig. 6.18b) and many plants. The rather special case of plants will be discussed in the next section.

The most important consequence of territoriality is population regulation. Territorial behaviour is closely allied to contest competition, and this, as we saw in section 2.4, leads to exactly compensating density-dependence. The contest nature of territoriality is demonstrated by the fact that when territory owners die, or are experimentally removed, their places are rapidly taken by newcomers. Thus, Krebs (1971) found that in great tit populations, vacated woodland territories were reoccupied by birds coming from

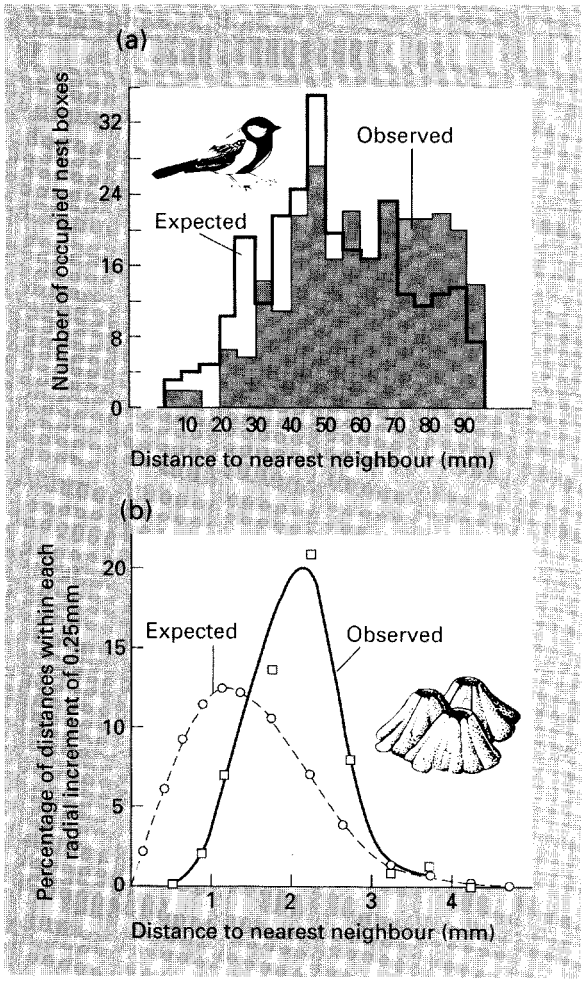


Fig. 6.18 Illustrations of territoriality. (a) Great tits (Krebs, 1971), and (b) barnacles (Crisp, 1961) spaced out more than would be expected from a random distribution on the available suitale habitats. (After Davies, 1978a.)

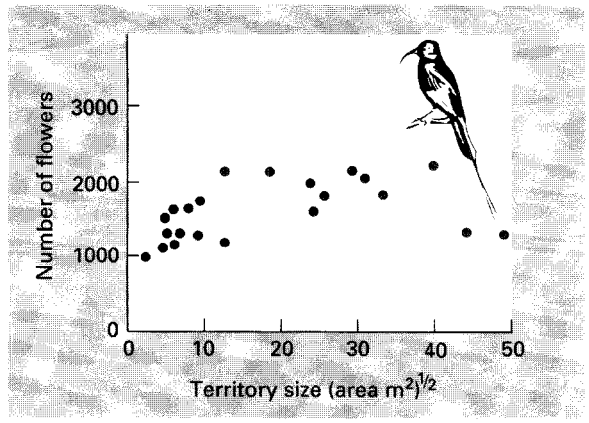


Fig. 6.19 Although the size of the territories of the golden winged sunbird *Nectarina reichenowi* varies enormously, each territory contains approximately the same number of *Leonotis* flowers (Gill & Wolf, 1975). (After Davies, 1978a.)

hedgerow territories where reproductive success was noticeable suboptimal: Watson (1967) found that with red grouse the replacements were non-territorial individuals living in flocks, which would not have bred, and would probably have died in the absence of a territory. In both cases, therefore, overall fecundity and population size were limited by territorial behaviour: by a 'contest' for a limited number of territories. Removal experiments have demonstrated similar phenomena in mammals (Healey, 1967; Carl, 1971), fish (Clarke, 1970), dragonflies (Moore, 1964), butterflies (Davies, 1978b) and limpets (Stimson, 1973). It must be realized, however, that the exact number of territories is usually somewhat indeterminate in any one year, and certainly varies from year to year depending on environmental conditions (Fig. 6.19); and it is, perhaps, for this reason that life-table analyses for the great tit and the red grouse fail to provide clear-cut evidence of density-dependence at the appropriate stage (Podoler & Rogers, 1975).

Wynne-Edwards (1962) felt that these regulatory consequences of territoriality must themselves be the root causes of territorial behaviour. He suggested that the selective advantage accrued to the population *as a whole*: that it was advantageous to the population not to over-exploit its resources. There are, however, powerful and fundamental reasons for rejecting this 'group selectionist' explanation—essentially, it stretches evolutionary theory beyond reasonable limits (Maynard Smith, 1976)—and Wynne-Edwards himself (Wynne-Edwards, 1977) has subsequently recognized these reasons and accepted the rejection of his ideas. Thus, if we wish to discover the ultimate cause of territoriality, we must search, within the realms of natural selection, for some advantage accruing to the *individual*.

It must be recognized that in assessing individual advantage, we must demonstrate not merely that there are benefits, but that these exceed the costs associated with territoriality. This has been done in the case of the golden-winged sunbirds examined in Fig. 6.19. Gill and Wolf (1975) demonstrated that although the size of territories may vary enormously, the nectar supply defended is suited to support an individual's daily energy requirements. They were able to measure the time that territory owners spent in various activities (including territory defence), and they showed that, as a result of the exclusion of other sunbirds, nectar levels per flower inside a territory were higher than in undefended flowers. Thus, Gill and Wolf found that territory owners could obtain their daily energy requirements relatively quickly, and that, overall, the energetic costs of territorial defence were easily offset by the benefits of the energy saved by a shortened daily foraging time.

A different type of individual advantage resulting from territoriality was demonstrated by Krebs (1971) for the great tit population of Wytham Woods (Fig. 6.20). There, the major predators of nestlings are weasels, *Mustela nivalis*, which may rob up to 50% of the nests in some years (Dunn, 1977). But, as Fig. 6.20 shows, the closer a nest is to another nest, the greater the chance of predation. Individual selection, therefore, favours the spacing out of nests: it pays each individual to be territorial.

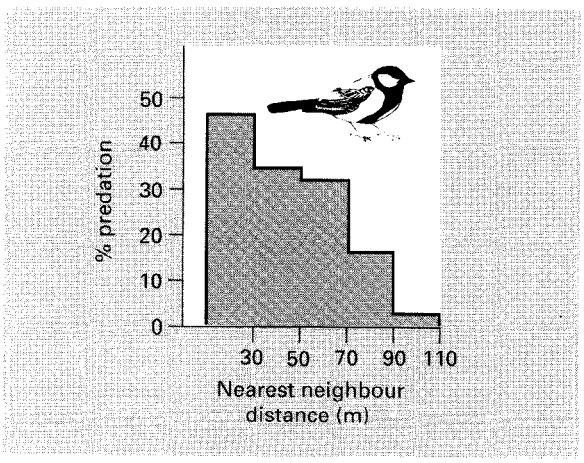


Fig. 6.20 The influence of territory size on the risk of predation in the great tit. (After Krebs, 1971.)

We can suggest then, from this very limited number of examples, that territoriality has evolved as a result of the advantages accruing to territorial individuals. But as an essentially independent *consequence* of this, there is competition approaching pure contest, and therefore powerful (though not, of course, absolute) regulation of populations. Figure 6.21 shows a key-factor analysis for the tawny owl population in Wytham Woods near Oxford (Southern, 1970) and demonstrates how territoriality can be translated into the currency of population dynamics through this method. The number of territory-holding adults changed very little over a 13-year period (see Fig. 5.1f), but there was a great deal of variation in the numbers attempting to breed (ranging from 22 pairs in 1959 to none in 1958). Failure to breed each year (k_1) was the key factor in this study. Poor small mammal years corresponded with years in which the owls did not attempt to breed. Prey availability was independent of owl density so k_1 *was* a density-independent factor. Losses outside the breeding season were, however, found to be density-dependent. Tawny owls are territorial all year round and these losses were primarily of young birds which were unable to establish territories and which consequently starved.

6.11 'Space capture' in plants

Although territoriality, as such, is not normally associated with plants, there is, in plants, a phenomenon which is at least analogous to territoriality. The phenomenon can be caricatured in the proverb: 'Possession is nine points of the law', and has been referred to and discussed by Harper (1977) as 'space capture'. In fact we have already discussed it briefly in section 2.5.2 as an explanation for skewed frequency distributions of plant weights.

Figure 6.22a shows that in experimental populations of the grass *Dactylis glomerata*, the comparatively low weights exhibited by late-emerging plants are lower than would be expected from the reduction in growing period alone (Ross & Harper, 1972). The reason for this is indicated by the data in Fig. 6.22b

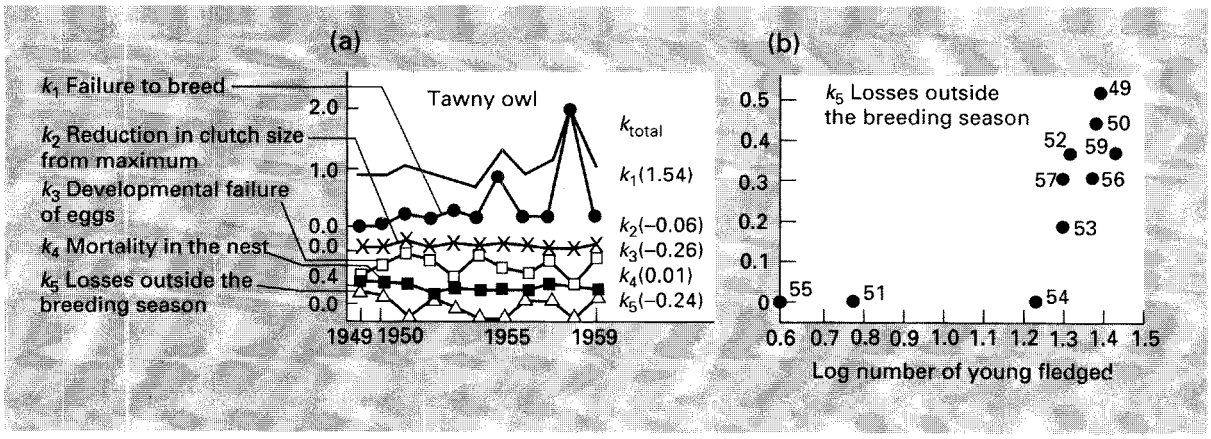


Fig. 6.21 Key-factor analysis for the tawny owl *Strix aluco*. (a) Total generation mortality and individual *k*-values plotted against time in years, with the regression coefficient of each individual *k*-value plotted against total generation mortality shown in brackets; k_1, failure to breed in poor small mammal years is the key factor. (b) k_5, losses outside the breeding season plotted against numbers of young fledged, illustrating a strongly density-dependent mortality. (After Southern, 1970.)

(Ross & Harper, 1972). Plants were grown from seed either under 'unrestricted' conditions: alone at the centre of a 7.4-cm diameter pot; or 'restricted' conditions: in a bare zone, 4.2 cm in diameter, surrounded by seeds sown at a density of 2.5 cm^{-2}. After initially growing at the same rate the 'restricted' plants grew at a slower rate than the 'unrestricted' plants, and maintained this difference for at least 3 weeks. It appears that the growth achieved by a plant, and thus the size and *fitness* it attains, is determined early in its life by the pre-emption or 'capture' of space (or the resources implied by that space). Space is then unavailable (or, at least, relatively unavailable) to other plants, and these grow more slowly and attain a lower fitness as a consequence.

Presumably, genetic predisposition to early emergence, and a chance association with favourable microsites, both play some part in determining which plants actually capture space. In either case, however, the result is to push competition towards the contest end of the scramble–contest continuum. The plants capture what is, in effect, a territory, and as a consequence there is a more exact regulation of plant numbers.

Essentially, the same phenomenon is shown by the herbaceous perennial *Anemone hepatica* in Fig. 6.23 (Tamm, 1956). Despite the crop of seedlings entering the population between 1943 and 1956, it is quite apparent that the most important factor determining which individuals were established in 1956 was whether or not they were established in 1943. Of the 30 specimens that had reached large or intermediate size by 1943, 28 survived until 1956, and some of these had ramified. By contrast, of the 112 plants that were either small in 1943 or appeared as seedlings subsequently, only 26 survived until 1956, and not one of these was sufficiently well established to have flowered.

Similar patterns are obvious from simple observations of tree populations. The survival-rate of the few established adults is high: that of the many seedlings and saplings is comparatively low. In all such cases it is clear that the major prerequisite for high (or indeed positive) fitness is the capture of space. We can think of this as the plant equivalent of animal territoriality, and the regulatory consequences are essentially the same.

The significance of pre-emptive space capture for the fitness of individual plants and the fact that plants are sedentary emphasizes the need to consider the spatial arrangement of plants in the regulation of plant populations. The structure of a population of a peren-

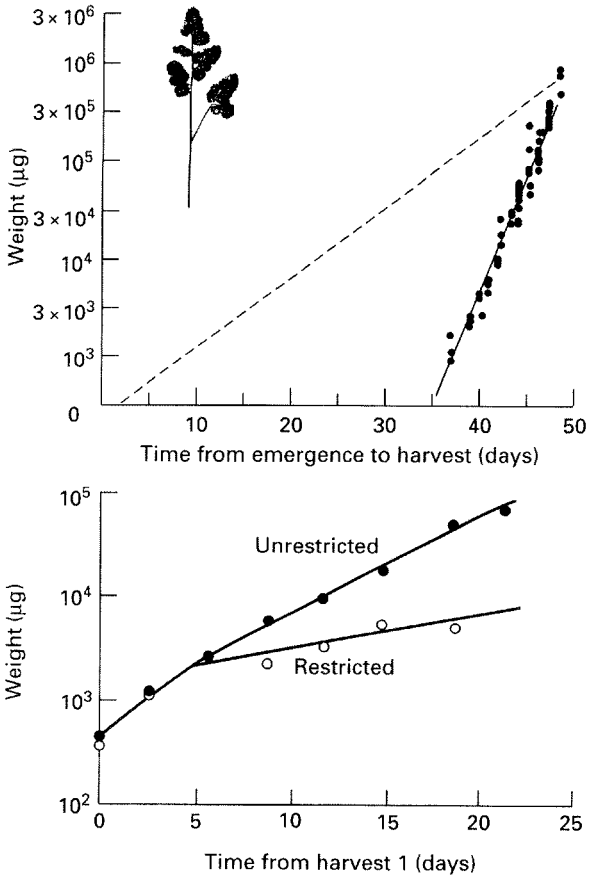


Fig. 6.22 (a) The influence of emergence time on the dry weight per plant of *Dactylis glomerata*. The dashed line shows the weights that would have been achieved had the weights of late emergers been attributable only to their reduced growth period. (b) The growth of *D. glomerata* seedlings with and without neighbours. (After Ross & Harper, 1972.) For further explanation, see text.

nial plant at any particular point in time will be a reflection of past phases of successful recruitment and the outcome of contest competition for resources, particularly light. The age- and size-structure arising from recruitment and the process of asymmetric competition will have a major bearing on the changing size distribution of individual plants (Hutchings, 1985). Recognition of the potential importance of the size of immediate neighbours to the fitness of any one individual has led to the use of *neighbourhood analyses*. In these the fitness components (yield and survivorship) of an individual are described in relation to the number of neighbouring plants occurring with increasing distance (Pacala & Silander, 1985). Whilst these approaches provide an important way of linking the performance of individuals to overall population performance, if only spatial interrelations are considered then considerable variation in the size of individuals is unaccounted for. As we have seen above, the relative time of emergence of a plant is crucial to an individual's success, and methods of analysis have still to be developed that adequately account for fitness in relation to both local spatial variation in neighbourhood density and time of plant 'birth' (Firbank & Watkinson, 1990).

Whilst considerable size inequalities may exist amongst individuals in plant populations for very many reasons, their influence on population dynamics may be not nearly so marked. Crawley (1993) has pointed out that the effects of size differences are often strongly mediated by plasticity in form and size as plants are inherently modular. The linear relationship between size and fecundity (see Fig. 2.11) means that the tendency for overcompensating density-dependence often evident in insect populations will be much reduced and in consequence plant populations are much less likely to show cyclical and chaotic dynamical behaviour. An apparent exception to this is *Erophila verna* which may exhibit cyclical dynamics (Silvertown & Lovett Doust, 1993).

6.12 Chaos in ecological systems

In section 3.4 we saw how time-lags, high reproductive rates and highly overcompensating density-dependence are capable of provoking many types of fluctuations in population density in single-species population models, without invoking any extrinsic cause. The most surprising type of population fluctuation that can be produced from such models is that which has been described as chaotic. One of the more surprising discoveries of population ecology in the 1970s was that many of the simple models of animal population dynamics were seen to display chaotic fluctuations (see May, 1974, 1976; May & Oster, 1976). Chaos has generated much interest because it

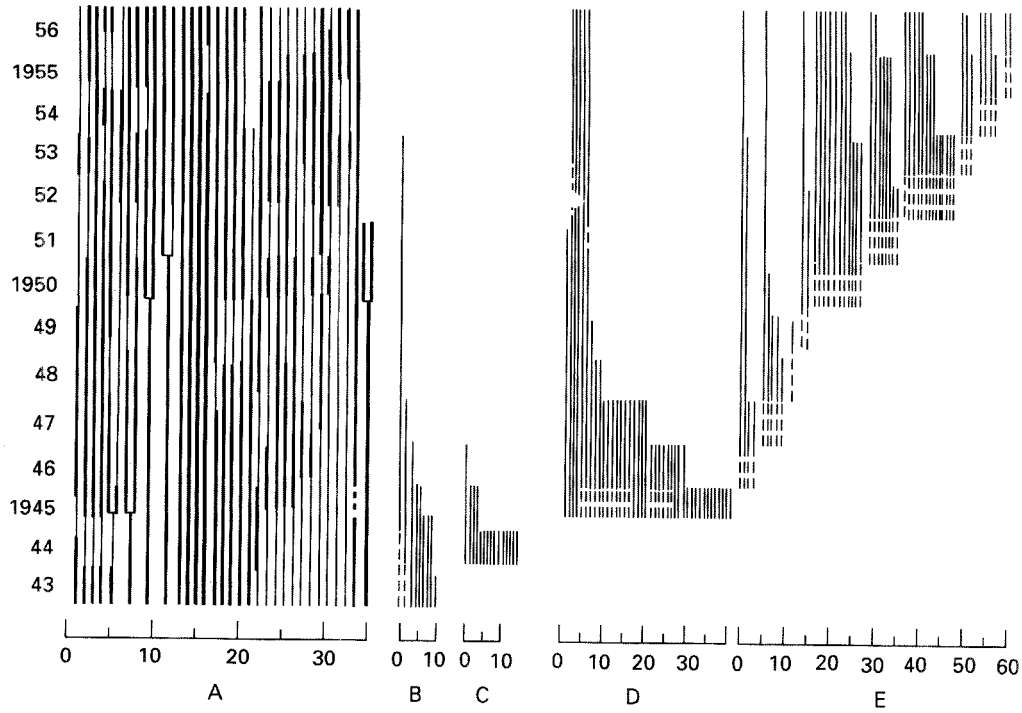


Fig. 6.23 The behaviour of *Anemone hepatica* in a forest. Each line represents one individual: straight for unramified ones; branched where the plant has ramified; bold where the plant flowered; and broken where the plant was not seen that year. Group A were alive and large in 1943; group B alive and small; group C appeared first in 1944; group D in 1945; and group E thereafter, presumably from seedlings. (From Tamm, 1956; after Harper, 1977.)

had previously been supposed that the irregular population fluctuations typically seen in real data must be due to random environmental fluctuations or sampling errors. That such fluctuations may arise in deterministic models in which all the parameter values are known exactly is quite remarkable. One of the aims of population ecology is to be able to understand the dynamics of populations to such an extent that future dynamics can be predicted. If chaotic behaviour prevents us from being able to do this with deterministic, simple models, then it will be extremely difficult to achieve it in real populations.

The discovery of the potential for chaos in population models stimulated a search for chaos in real populations. This search has progressed along three fronts. The first involves fitting population models capable of predicting a range of dynamics from stable to chaotic depending on the values of one or more parameters. The second is concerned with the examination of data from laboratory populations, usually of insects, that have been maintained over a large number of generations. The third involves the development of new analytical techniques from non-linear mathematics designed to detect chaos in time-series data. We shall consider each of these techniques below.

Hassell *et al.* (1976) were the first to fit models to field data in this context. They estimated the parameters of a simple single-species discrete-generation model (equation 3.4) capable of showing chaos, using 28 sets of insect population data (24 from the field and four from the laboratory). Their results are shown in Fig. 6.24 (see also section 3.4). With two exceptions all the field life-table data fell in the region of monotonic damping, with a stable equilibrium point. Of the remaining field studies, one fell in the region of

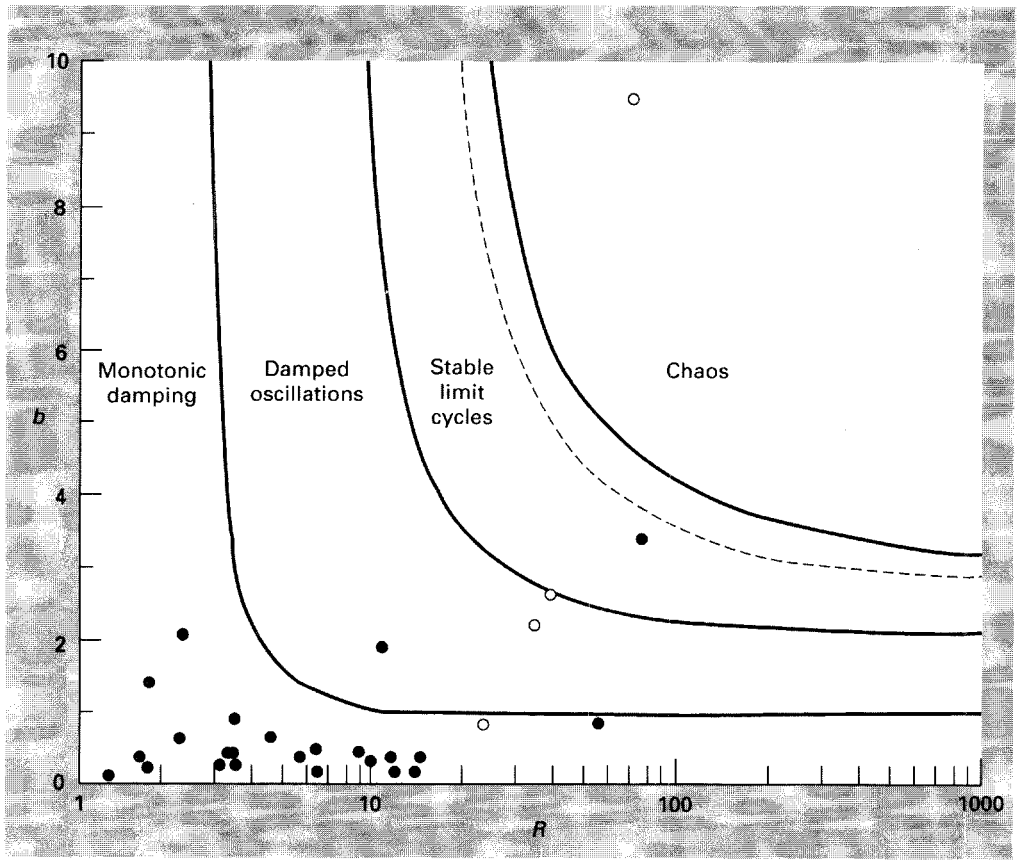


Fig. 6.24 Stability boundaries between the density-dependent parameter, b, and population growth rate, R, from equation 3.4, together with estimated parameters from field (●) and laboratory (○) studies. (After Hassell *et al.*, 1976.)

damped oscillations and the other in the region of stable limit cycles. This last study was of the Colorado potato beetle *Leptinotarsa decemlineata* a well-known outbreak pest in present day agroecosystems (see section 6.6). Of the four studies of laboratory populations, one fell in the region of monotonic damping, two fell in the region of damped oscillations, and the fourth fell in the chaotic region. Figure 6.24 could be interpreted as indicating a tendency for natural populations to have stable equilibria while laboratory populations show cyclic or chaotic behaviour. There were, however, a number of important assumptions implicit in this study, many of which the authors themselves point out. The most serious of these is that there are no truly single-species populations in the real world, and it is unclear how valid it is to abstract the dynamics of any population from its natural community to a single-species population model. In addition the use of a single-species model biases the results in favour of stability since complex dynamics are more likely in complex systems. We have already seen how delayed density-dependence, one phenomenon likely to arise as a result of biotic interactions, is found in many insect populations (Turchin, 1990) but remained obscure from those looking for density-dependence from life-table studies (see section 6.7.1).

A number of other workers have looked for evidence of chaos in laboratory populations, mostly of *Drosophila* (e.g. Thomas *et al.*, 1980; Mueller & Ayala, 1981), but failed to find it. However, by the late 1980s the laboratory approach seemed to be more promising

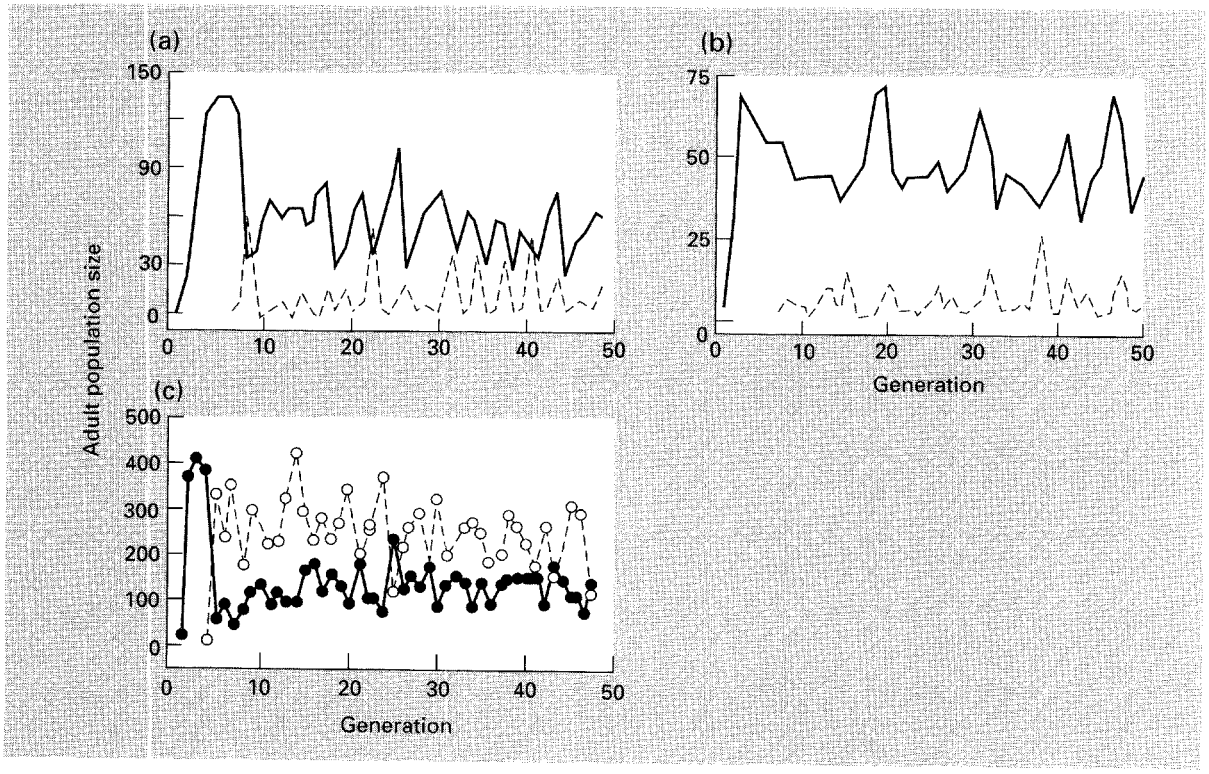


Fig. 6.25 (a) Predicted dynamics for the interaction between *Callosobruchus chinensis* (——) and the pteromalid parasitoid *Lariophagus distinguendus* (– – –). The stimulations were started with two adult hosts on day 1 and 0.02 parasitoids were added on day 17 of the seventh generation. (b) As for (a), but for *Callosobruchus maculatus* and *Lariophagus distinguendus*. (c) Observed dynamics of populations of *Callosobruchus chinensis* (●) and pteromalid *Anisopteromalus calandrae* (○) from the laboratory system of Utida (1950). (After Bellows & Hassell, 1988.)

with the discovery of chaos in a series of population models, particularly host–parasitoid models, with parameters within the range of biological realism. For example, Bellows and Hassell (1988) investigated an age-structued host–parasitoid model which exhibited intriguing irregular fluctuations using experimentally determined parameter values (Fig. 6.25a & 6.25b). The irregular cycles seen in a study of Utida (1950) of a similar host–parasitoid system, the bruchid beetle *Callosobruchus chinensis* and its pteromalid parasitoid *Anisopteromalus calandrae* were qualitatively so similar to the results of the simulation model of Bellows and Hassell that they, too, may arise from the internal dynamics of the interaction (Fig. 6.25c).

The third approach in the search for chaos in real populations has been the development of techniques for distinguishing chaos from random noise in the analysis of time-series. One such scheme is that of Sugihara and May (1990), who used a library of past patterns in a time-series to make short-term predictions about future patterns. By then plotting the correlation coefficient of prediction and actual trajectories against prediction time interval they considered it possible to make the distinction between dynamical chaos and measurement error. This is based on one of the 'signatures' of chaos, namely that in a chaotic system, the accuracy of forecast falls off with prediction time, whereas in a system that is merely 'noisy' the forecasting accuracy is roughly independent of the prediction interval. These new mathematical techniques are particularly demanding of data and there are few biological data sets of sufficient quality and

length to be suitable for the application of the techniques. However, data from childhood diseases, notably measles and chicken pox have been analysed by Sugihara and May (1990). The non-linear forecasting technique seems to indicate that prediction error in measles occurs in a manner consistent with chaotic dynamics but that this is not the case with chicken pox where prediction error is characteristic of pure additive noise (actually superimposed on a seasonal cycle). However, the conclusions of the childhood diseases analysis and the technique itself have been challenged by Hastings *et al.* (1993).

Another approach to detecting chaos in time-series data is that of Turchin and Taylor (1992). Their approach in some ways follows that of Hassell *et al.* (1976), but deals with multi-species systems by including in the model delayed population density, which reflects the interactions with other species. Unlike Hassell *et al.* (1976), Turchin and Taylor did not specify a particular equation, but fitted a response surface, the logarithm of yearly population change, as a function of lagged population densities (see the Turchin & Taylor paper for details of the technique). Turchin and Taylor analysed time-series data for 14 insect and 22 vertebrate populations. Their results were different from those of Hassell *et al.* (1976). They found exponentially stable point equilibria in only three out of 14 insect populations, compared with 21 out of 24 field studies reported by Hassell *et al.* (1976). The remaining populations displayed unregulated behaviour (one), damped oscillations (six), limit cycles (one), quasi-periodic oscillations (two) and chaos (one). The vertebrate data exhibited a similar range of behaviours, though none was chaotic. Thus the conclusion that Turchin and Taylor reached was that the complete spectrum of dynamical behaviours ranging from exponential stability to chaos can be found in natural systems. (The single case of chaos has, however, been questioned by Perry *et al.*, 1993 on the basis of more extensive data.) Although to some extent the same criticisms can be levelled at this work as that of Hassell *et al.*, in that they assume a particular type of underlying model, the model and fitting techniques are more robust than those used in the earlier study. The significant similarity between the two studies is that both suggest that chaos is rare in field populations of insects.

One study which adopts both the first and third approaches to investigating chaos, fitting population models and detection from time-series, is that of Hanski *et al.* (1993), who explored the population oscillations of microtine rodents in boreal and Arctic regions. Their population model was a predator–prey model with seasonality in which the predators were mustelids. The model was able to generate stable dynamics, limit cycles and chaos and when parameterized with field data predicted dynamics that closely resemble the dynamics of boreal rodent populations. The analysis of extensive time-series data of microtine rodents in Fennoscandia was achieved by using the technique of non-linear analysis developed by Turchin and Taylor. Both the model prediction and the observed dynamics are chaotic. This result suggests that the multi-annual oscillations of rodent populations in Fennoscandia are due to delayed density-dependence imposed by mustelid predators, and they are chaotic.

How to distinguish chaos from measurement error and how to find it in natural populations are some way from being resolved. The search for chaos is likely to be a particularly vigorous area for research in the 1990s (see Godfray *et al.*, 1991 and Hastings *et al.*, 1993 for reviews).